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PITFALLS OF GENERAL SURGERY

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As one lives with a surgical practice he becomes aware of certain recurring problems in diagnosis and treatment that stand out in his memory because, with the solution of each problem, the surgeon is likely to have acquired another painful experience. These problems seem to develop according to a pattern or plan designed by a diabolic power intent on deceiving the surgeon. A patient following such a pattern is likely to have an innocuous onset of easily recognized signs and symptoms that suggests an early diagnosis and an accepted form of treatment. However, following a reasonable course of action such a patient's condition is likely to go from bad to worse, and unless the surgeon recognizes the trap into which he has fallen the consequences may become disastrous.

These traps or pitfalls come in a wide variety of forms and may be found in all branches of medicine. They have one common denominator, namely, an early, easy, erroneous diagnosis based on a clinical finding that heretofore has been completely reliable. The reliability of a given sign in diagnosing a given disease process or the infallibility of a certain symptom in leading to a correct diagnosis also measures the danger of the sign or symptom when it turns up as a finding in one of the pitfalls we are discussing. For example, the experience that incision and drainage when fluctuation is present brings prompt relief to an abscess, whereas an incision into an inflamed area before fluctuation develops may be harmful, makes the surgeon believe he should wait for fluctuation before incising an area in order to produce drainage. However, if one waits until fluctuation develops in the neck or the parotid gland or in the closed space of a terminal phalanx, he has waited much too long and is guilty of making an error of omission.

An uncertain or erroneous diagnosis may cause trouble in either of two ways. It may lull the surgeon into a false sense of security which results in an error of omission. As a matter of fact, a surgeon should reconsider whenever he says to

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the patient, "Let's watch it." Or, worse yet, the false diagnosis may be a frightening one which causes the surgeon to fear he is losing valuable time and therefore causes him to rush into an error of commission—the "eager beaver" syndrome. No matter which form the trap takes the patient suffers, because the worst possible situation for a patient is to find himself cared for by a surgeon who is satisfied with an erroneous diagnosis.

Errors of omission usually result in losing valuable time. As a result the lesion progresses to a less favorable state or the patient is allowed to die without the medical or surgical help that might have saved him. Every time a surgeon uses the phrase, "Let's watch it," he is treading on thin ice. In the first place the phrase means to the patient the lesion is definitely not dangerous so, hoping for the best, the patient is likely not to return for a year or two. In the second place the only excuse for watching a lesion is the belief that the situation will either remain the same or improve with the passage of time. Tumors rarely improve; they usually enlarge, and their late removal is more difficult than their early removal. It is so much better to solve any given problem immediately. In general, solid tumors anywhere and cystic masses in the ovary as large as a tennis ball should be removed without delay. At times it is difficult to be sure whether a given mass is cystic or solid, but the simple procedure of aspiration usually solves this problem safely. Of course it is not wise to aspirate intra-abdominal cysts blindly, but in the neck and in the breast this procedure is quite safe and adds greatly to the accuracy of diagnosis. Also in the case of a breast cyst, aspiration may result in the cure of the cyst.

There are a few physiological swellings of breasts and ovaries that subside with passage of the menstrual period. Waiting through one cycle to check the changes due to the cycle is permissible, but waiting longer than one month is usually not advisable. If one decides to watch such a lesion he should have a plan and set a deadline at which time the problem will be re-evaluated. It would be so much wiser to tell the patient, "This area is not normal. It could be the sort of tissue that changes with the menstrual periods, or it could be the sort of tissue that should be removed. Therefore re-examination after the next menstrual period should solve the problem. If the area has returned to normal we will both be happy; if not, we can arrange to remove the area for further analysis."

One of the most tragic errors of omission is the error of considering a given process to be a far advanced malignant lesion, probably incurable; therefore no therapy is attempted. Occasionally such lesions are benign from the onset, and proper treatment could have saved the patient any time prior to the last few days of his life. Examples of such lesions come to mind immediately: the jaundiced patient considered to be suffering with cancer in the liver, but in reality having only a stone in the common duct; the large gastric ulcer surrounded by a granuloma in the mesentery of the colon or omentum, abandoned because the lesion suggested inoperable gastric cancer; the large lymphosarcoma or leiomyosarcoma of the stomach confused with stomach cancer; and diverticulitis of the colon with granuloma and fistula formation confused with far advanced colonic

cancer. These and many other lesions can not be definitely diagnosed without microscopic proof of the diagnosis. It is my opinion that only rarely should a patient be permitted to die with a diagnosis of hopeless cancer unless that diagnosis is confirmed by a positive biopsy examination. A small biopsy operation can be carried out with local anesthesia with very little risk to the patient and with very little expense. Its value is tremendous. The accurate diagnosis makes subsequent management of the patient far easier, especially when the question of terminal deep sedation arises. The authority of the microscopic examination also makes management of the patient's relatives easier. They are less likely to fly off to try will-of-the-wisp treatments if the surgeon can speak definitely and surely of the microscopic diagnosis; whereas, if there is no biopsy examination the surgeon must be a little vague when asked, "Are you sure it's cancer?" A vague answer allows the family to hope the surgeon is wrong in his bad prognosis and this hope spurs them to seek anyone who will promise a better prognosis. Quacks will promise anything for a price.

While considering errors of omission it should be pointed out that one serious error is the failure to check hearsay reports before the operation is started. The operative findings and pathological reports of previous operations should be carefully checked, not just taken on the patient's or interne's memory of these findings. Also serology reports, the matter of drug sensitivity, and the history of dangerous drug therapy all should be checked as far as possible. It might be worth while to be sure the urine sent for A Z test was the patient's urine and not her husband's urine.

Last, I would like to mention a common omission we all tend to make because we all dislike talking about unpleasant matters. This is the failure to prepare the patient for bad news. If we lead up to the bad news, mention the possibility for a day or so before making the actual statement, then the patient has had a little time to adjust, to prepare himself, to rationalize, so that he can take the bad news without losing his composure. If you tell a patient he may require colostomy, or an amputation, or additional operations, and then wait a day or so, he will accept the statement that one of these events is inevitable much better than he would had you not given him the chance to think it over as a possibility. This same technic is useful when it is decided to tell a patient his biopsy report is "malignant." Failure to prepare a patient for bad news is cruel, and may result in the patient's changing doctors.

There is a definite emotional pitfall peculiar to the practice of surgery. Since a surgeon treats his patients chiefly by means of some sort of operation, he is likely to approach each new problem wondering what sort of operation will be best for this patient. As a matter of fact, this attitude is expected of him. The family of the patient, the internist, and usually the house staff are all pushing the surgeon toward the operating room. At times the lack of a positive diagnosis plus the fear that valuable time is being lost sets the scene for an unnecessary or ill advised operation. Every surgeon has felt this pressure and most of us have yielded to the urge to "do something" and carried out an operation we later regretted. For example, operations to relieve vague pains are likely to be

unsuccessful. Operations to release supposed adhesions rarely do much good unless there is associated intestinal obstruction. Operations made to save a patient's face, to justify his or her years of complaining, are doomed to failure. In such circumstances even if you are told that if you don't "suspend this uterus" or "explore this abdomen someone else will do it," my advice is to let someone else do it, because usually everyone connected with such a procedure comes out a loser. No one can make you operate upon a patient. If you don't think an operation is really best for the patient, give up the case. Usually there is no great hurry. My rule is to operate only when a patient is getting worse or failing to improve after careful medical attention. I rarely operate when the diagnosis is uncertain and the patient seems to be improving.

When we consider surgical pitfalls that lead to errors of commission we find that an erroneous diagnosis is usually the first step in the wrong direction. Specific examples of the traps into which the "eager beaver" is likely to fall as he makes an error of commission are numerous: for example, re-operation in the early postoperative period for release of adhesions producing partial intestinal obstruction. Such adhesions are usually caused by fibrin which will absorb if the gaseous distention is controlled by a long nasal suction tube of the Miller-Abbott type. Early operation for appendix abscess is likely to do more harm than good. About half of the masses in the right lower quadrant diagnosed as appendix abscess will either absorb or drain into the caecum. It is much wiser to wait up several days before deciding to operate upon these patients, especially if the mass has been present only a day or so. In general it is not advisable to probe or open a hematoma with a hemostat until it has been present 8 to 10 days and is in a liquid state, usually indicated by a black soft blister in the scar itself. This is especially true following thyroidectomy. Such hematomas should either be left alone for several days or, if pressure symptoms demand early action, the wound should be opened widely and the jelly-like clot evacuated.

An especially dangerous pitfall occurs when a poor-risk patient must be operated upon. The unwary surgeon in his eagerness to cure every patient may choose the type of operation unwisely. For example, cholecystostomy at times is a better operation than cholecystectomy, and simple mastectomy with local anesthesia for breast cancer a better operation than radical mastectomy carried out with 2 or 3 hours of general anesthesia. Gastroenterostomy with or without vagectomy is a better choice for peptic ulcer than partial gastrectomy in certain elderly or poor-risk patients. I am not trying to say a Ford is better than a Lincoln. What I want to emphasize is that just as everyone's bank account isn't able to afford a Lincoln, so every patient's stamina and reserves aren't able to afford the more dangerous perfect operation. The poor-risk patient will get farther with a lesser operation he can take safely than with a more extensive operation that may prove too much for him.

No one has a higher opinion of the biopsy operation than I, but at times I believe that surgeons make meddlesome and dangerous use of this operation. It is my opinion that whenever we cut into a cancer we spread it throughout the wound just as we would infect the large wound made to excise an abscess. In

the case of the patient with far advanced cancer where cure is out of the question, this spread is not so important, but in the patient who can be cured by block dissection it is probably better never to cut into the growth or across its paths of spread. If such biopsy is absolutely necessary the wound in the cancer should be cauterized, packed and closed and all contaminated instruments discarded. Also the curative procedure should follow as soon as possible after the incision into the cancer. This brings us to the question of when and where the biopsy operation should be done. I believe the surgeon who is equipped to carry out the major procedure should be the one to make the biopsy exploration so that prompt removal of the growth can be carried out if the pathologist reports cancer. The pitfall here is: who is qualified to carry out the definitive procedure? This is a matter of conscience. It is so easy to read a report, to understand it completely and then feel that you can duplicate the author's results. We all know this isn't so. We must realize our limits and operate just within or just beyond those limits. It isn't fair to yourself or your patient for you to get in far beyond your depth. You never lose a patient's respect when you refer him to someone who can carry out a certain procedure better than you can.

It would not be helpful to enumerate all the disease processes that have been misdiagnosed or mismanaged in the course of a lifetime. The author would, however, in closing like to draw attention to two specific disorders that have caused him embarrassment on more than one occasion. The first is mechanical obstruction of the small bowel where the bowel, due to long standing distention, progresses to a state of paralysis and thus may be confused with paralytic ileus. To add to the confusion, paralytic ileus most often due to peritonitis may in the late stages progress to mechanical intestinal obstruction. This is brought about by the presence of fibrin which tends to immobilize heavy fluid-filled loops of bowel in the pelvis. Such loops become kinked and twisted and produce mechanical obstruction as effectively as any fibrous adhesive band could. The problem is further complicated by the fact that the treatment of the two diseases, mechanical intestinal obstruction and paralytic ileus, is exactly opposite, since early operation is desirable for the former and extremely undesirable for the latter. Because early certain diagnosis of mechanical intestinal obstruction is often impossible, it follows that we always must be suspicious that mechanical obstruction may be present in any patient with intestinal cramps, especially if auscultation of the bowel shows increased bowel sounds synchronous with the height of the pain. The pain-sound relationship is not present in the cramps of kidney stone, gallbladder colic, twisted ovary or pancreatitis. It can even be elicited, but with difficulty, when mechanical obstruction develops late in paralytic ileus or in the postoperative period. Here the observer may have to listen for long periods before the tell-tale tinkle is heard at the same time a feeble cramp occurs. Once this relation of pain to bowel noise is established, the burden of proof lies with the one who says mechanical obstruction is not present and that early operation is not indicated. Far too many patients are watched to death when mechanical intestinal obstruction reaches the paralytic stage, or when paralytic ileus develops a mechanical obstruction component. All that can save

such a patient is operation with release of the obstruction and operative decompression of the small bowel by means of one or more enterostomy openings and suction to remove the obstructed small bowel gas and fluid.

The other disorder that has caused the author more than just a little trouble more times than just occasionally is the silent gallstone. Here we have a ready diagnosis for abdominal pain; in fact, there are illustrations in the form of X-ray films that come with the patient. However, it is always possible that the pain that brings the patient in asking for an operation is caused by a disease in some nearby organ, and the easily visualized stone is as silent now as it has been for many years. Removal of the gallbladder and such a stone will not cure the patient's pain, and it might be extremely dangerous if the real cause of the pain happened to be coronary occlusion or hepatitis. As in the other surgical pitfalls awareness of their presence is our greatest protection, so with the silent gallstone we must be especially slow to advise its removal when it seems recently to have ceased to be silent and to have become symptomatic. A patient with symptoms thought to be caused by formerly silent gallstones should be studied extremely carefully with cardiograms, gastrointestinal X-rays, barium enemas, and stool and blood examinations in order to avoid overlooking the real cause for the symptoms that made him ask for removal of a stone he has been willing to carry for years.

And so it goes. There is no way that we can avoid facing the many pitfalls that will be encountered in the practice of surgery. A surgeon's life becomes easier and his patient's life safer as the surgeon becomes aware of the many pitfalls in diagnosis and suspicious of even his favorite signs and symptoms. It is the author's fond hope that this discussion might transfer his experience to others so that they would avoid many if not all the pitfalls he has mentioned. But human nature being as it is, and surgeons being as they are, and since there is rarely any learning without pain, it follows that we all will probably be scrambling out of pitfalls the rest of our lives. It is to be hoped, however, that we start scrambling out before we get very deep in the trap and before we qualify for the title "eager beaver" or membership in the "let's watch it" society.

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BENIGN GIANT DUODENAL ULCER*

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A benign giant ulcer of the duodenum is "benign" only in the sense that it is not malignant. Previously reported cases indicate that this is a rare and quite lethal form of peptic ulcer. The condition has proved to be almost uniformly fatal unless an accurate diagnosis was made and surgical treatment instituted. Unfortunately, the majority of these ulcers in the past have been recognized only at autopsy. Experiences on the surgical service of the University of Arkansas at the University and State Hospitals have led us to believe that giant ulcers in this location are not as rare as the literature would indicate. Eight patients with such lesions were encountered from 1950 to 1957. It is the purpose of this paper to relate these experiences. Inasmuch as the diagnosis must rest largely on a high degree of suspicion in many instances, an increased awareness of this picture on the part of the medical profession is necessary if more frequent diagnosis and effective treatment is to be accomplished. The great difficulty lies in the fact that routine roentgenologic examination of the upper gastrointestinal tract by barium meal may appear normal or only slightly abnormal when in reality a giant peptic ulcer is present in the duodenum.

HISTORIC

The first report in the American literature concerning benign giant duodenal ulcers was that of Freedman and Goehring[5] who reported 2 cases in 1940. In reviewing the accumulated literature, they gave Brdiczka[1], who in 1931 reported 3 cases, credit for first describing the condition and for making the first ante mortem diagnosis. In the interim, they found one other report of 4 cases by Knutsson[7] in 1932. One of these latter proved to be a carcinoma. Single case reports have been added to the literature by Elkin[3] in 1941, Kahlstrom[6] in 1944, Evashwick[4] in 1951, Bullock and Snyder[2] in 1952, and Ruby[9] in 1953. Of these 13 reported cases, a correct diagnosis was made only in 3 patients prior to surgical exploration or autopsy. The correct diagnosis was made in 4 patients at operation and in 6 patients at autopsy. The primary cause of death was hemorrhage or its complications in the latter group of patients.

DEFIN TION AND CRITERIA OF DIAGNOSIS

Uniform criteria have not been used by all authors in defining a giant sized ulcer. Those 2 cm. and over have usually been placed in this category. Based upon the fact that MacCarty's[8] study of 425 excised duodenal ulcers showed none to exceed 2.5 cm., Bullock and Snyder suggest the following criteria be

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filled in making a diagnosis, "that only ulcers greater than 2.5 cm. be considered as giant ulcers. The ulcer should be located in the first part of the duodenum, accurately measured and histologically studied either at surgical exploration, extirpation or autopsy. If the diagnosis is made by roentgenographic methods or no histologic examination is made, a 3 year clinical follow-up should be made in order to avoid including carcinomas of the duodenum". In reviewing previous reports, they could find only 5 cases including their own which met these criteria. The cases of Brdiczka who originally described the condition as well as Knutsson and Freedman and Goehring were eliminated. While 7 of our 8 cases meet these criteria, one does not because accurate measurements were not taken. In this case however, an estimate of 4 or 5 cm. was given by the surgeon and the description of the pathologic picture leaves no doubt that an ulcer larger than 2.5 cm. was present. It further seems more important to emphasize that these lesions are more common than the literature indicates rather than making them appear even more rare. Otherwise the possibility will not be kept in mind. We prefer to accept all of the 13 previously reported cases and wish to add 8 additional ones of our own.

CASE REPORTS

Case 1. G. M., a 38 year old white man, was admitted to the University Hospital on Nov. 3, 1953, following a massive hematemesis which resulted in the patient's fainting. The patient had suffered from typical ulcer symptoms for 8 years. A duodenal ulcer had been demonstrated by his local physician several years previously on x-ray examination. For the preceding 2 weeks, there had been an exacerbation of symptoms including intermittent tarry stools. Physical examination disclosed an obvious anemia, impending shock and mild epigastric tenderness. The red blood cell count was 2.56, hematocrit 20 per cent and hemoglobin 7.5 gm. Shortly after admission, the patient went into shock which responded to whole blood transfusions, but bleeding continued and the patient would not stabilize. After 4,000 cc. of blood was administered and 12 hours after admission, the patient was transported to the operating room where it was necessary to give him an additional 2,500 cc. of blood before an emergency operation could be undertaken. At laparotomy the duodenal area presented as an inflammatory mass 8 cm. in diameter which at first was thought to represent a neoplasm. However, further dissection revealed a giant ulcer 4 cm. in diameter in the first part of the duodenum which had destroyed most of the lateral walls and penetrated into the pancreas. Fragments of the bleeding pancreaticoduodenal artery were ligated in the base of the ulcer. The duodenum was trimmed away from the ulcer bed and a Billroth II type of gastric resection performed (fig. 1). It was impossible to close the duodenal stump because of the inflammatory reaction and proximity of the ampulla of Vater. Instead a catheter duodenostomy was substituted. The patient made an uneventful recovery and had no ulcer symptoms when last observed 6 months following operation. Microscopic sections of the duodenum disclosed the border of a benign peptic ulcer.

Case 2. A. T., a 71 year old white man, had been a custodial case at the State Hospital for Nervous Diseases since 1930. In 1947 an abdominoperineal resection had been performed for carcinoma of the rectum. No recurrence of the malignancy developed. On the fifth of September 1950, without any previous symptoms, the patient suddenly began to vomit large amounts of blood and died within a few minutes. At autopsy the stomach and intestines were filled with blood. In the duodenum just proximal to the papilla of Vater, there was a large penetrating ulcer 2.5 by 5 cm. in size which had destroyed the duodenal wall and eroded into the pancreas (fig. 2). A calcified and eroded pancreaticoduodenal artery

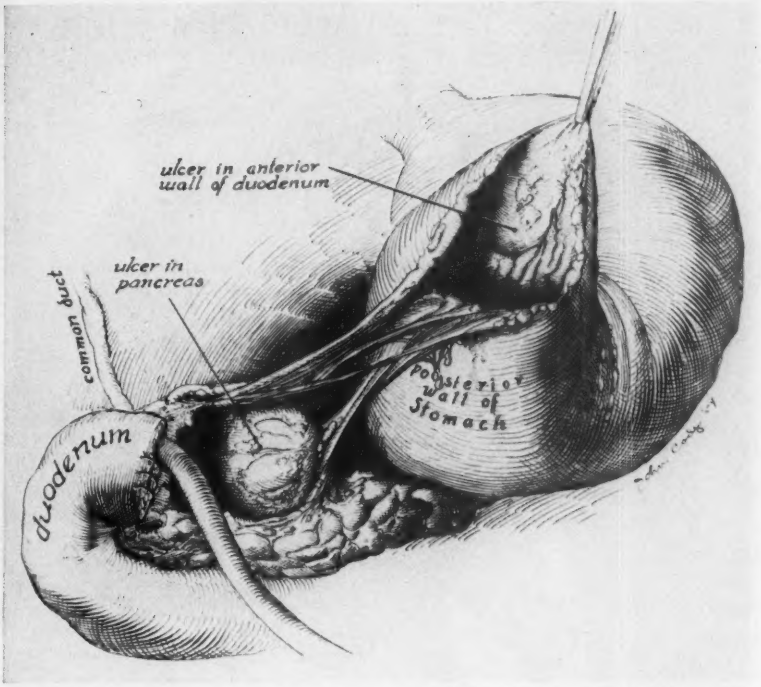

FIG. 1. Artist's concept of the giant ulcer and catheter duodenostomy at operation in Case 1.

was present in the base of the ulcer to account for the sudden exsanguinating hemorrhage. Microscopic sections showed a benign peptic ulcer.

Case 3. H. B., a 51 year old white man, was admitted to the University Hospital emergency room on June 19, 1955 in profound shock following several episodes of vomiting blood in the 2 hours preceding admission. For the previous 3 months, the patient had experienced burning epigastric pain and was under treatment by his physician for peptic ulcer. No x-ray studies had been made. Medical advice had been poorly followed as the patient was uncooperative and a chronic alcoholic. After receiving 2,000 cc. of whole blood, the patient's blood pressure and pulse stabilized at normal levels and an emergency gastrointestinal series was obtained. Large filling defects presumed to be the result of blood clots were noted throughout the body of the stomach and in the duodenal bulb. The bulb was consistently deformed and there was thought to be a constant barium fleck near the base of the bulb representing an active ulcer crater (fig. 3). The patient was admitted on the medical service and started on a Sippy diet ulcer regime. Three days after admission, the patient again went into shock following massive hematemesis. After controlling shock with multiple blood transfusions, an emergency laparotomy was performed. A deeply eroding posterior ulcer 2.5 by 2.8 cm. in size was found in the first portion of the duodenum. The pancreaticoduodenal artery was eroded and actively bleeding in the ulcer bed. There were two small shallow acute ulcers in the prepyloric area of the stomach which were not bleeding. The duodenum was dissected from the ulcer bed and a Billroth II type gastric resection performed. Closure of the duodenal stump was accomplished with difficulty and this area was

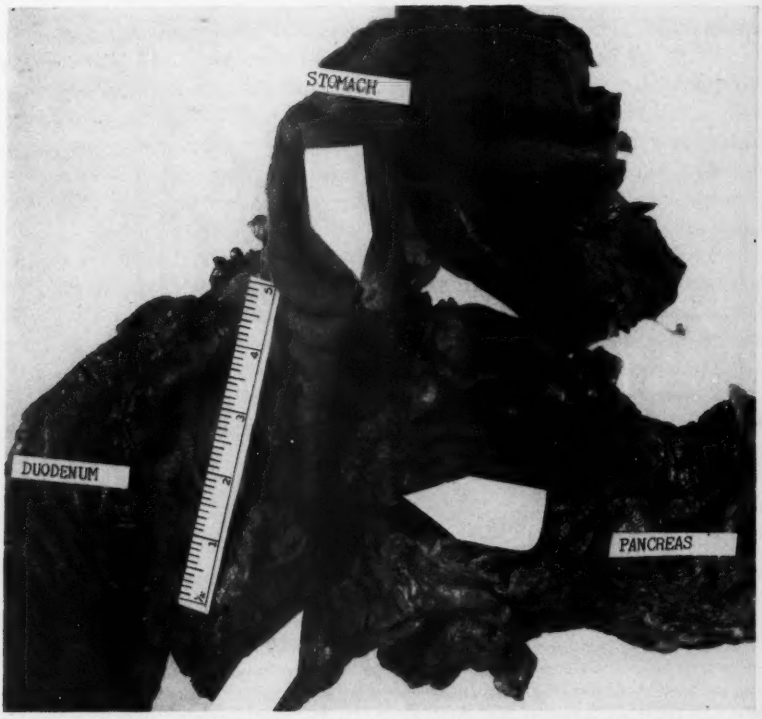

FIG. 2. Autopsy specimen Case 2. Note the eroded pancreaticoduodenal artery in the base of the ulcer.

drained. Microscopic studies of the excised duodenum revealed a benign peptic ulcer. The postoperative course was stormy, the patient developing an aspiration pneumonia and a low salt syndrome. These were successfully combated however and the patient was discharged 10 days following operation. When last examined 1 month later in the out-patient clinic, the patient was asymptomatic.

Case 4. E. M., a 45 year old Negro male deaf mute, was admitted to University Hospital on Sept. 23, 1955 because of persistent nausea and vomiting. For 5 years, the patient had suffered from intermittent epigastric pain which had become more severe the preceding 3 months and associated with coffee ground vomitus and tarry stools. There had been a 22 pound weight loss during the recent exacerbation. At the time of hospitalization, no ingested food could be retained and the patient presented a classical picture of malnutrition, hyponatremia, hypochloremia and alkalosis. The patient was placed on gastric suction and given replacement therapy parenterally.

Roentgenologic studies of the gastrointestinal tract demonstrated an almost complete obstruction of the stomach in the pyloric or prepyloric area. The stomach was dilated and irregular in the adjacent portion. Distal to the obstruction, there was a collection of barium which appeared to be the duodenal bulb. It was noted however to change very little in contour. The possibility was entertained that the collection represented a giant duodenal ulcer although the most likely diagnosis was considered to be an antral carcinoma (fig. 4).

At operation there was a marked periduodenitis associated with adhesions over the upper abdomen. A giant ulcer 4 to 5 cm. in diameter occupied the posterior aspect of the

FIG. 3. Ulcer in Case 3 which appears essentially as a normal duodenal bulb with filling defects interpreted as blood clots with a possible ulcer niche.

first portion of the duodenum. This had penetrated into the head of the pancreas and extended superiorly over the hepatoduodenal ligament to penetrate into the posterior aspect of the liver. Distally its edge extended nearly to the ampulla of Vater. The entire posterior wall and most of the superior wall of the duodenum had been destroyed. Tedious dissection was required to ascertain the pathology and remove the duodenum from the periphery of the ulcer crater. A Billroth II type gastric resection was performed. The usual layer closure of the duodenal stump was impossible because the posterior wall at the distal edge of the ulcer was in too close proximity to the ampulla of Vater. Instead the anterior wall of the duodenum was sutured to the margin of the distal portion of the ulcer and ulcer bed after the method of Nissen. The duodenal stump was drained. Microscopic examination of the duodenum disclosed the edge of a benign peptic ulcer.

Postoperatively pancreatic juice began to drain from the drainage site on the fifth day. Sump drainage was instituted. The patient progressed to a full liquid diet but his appetite

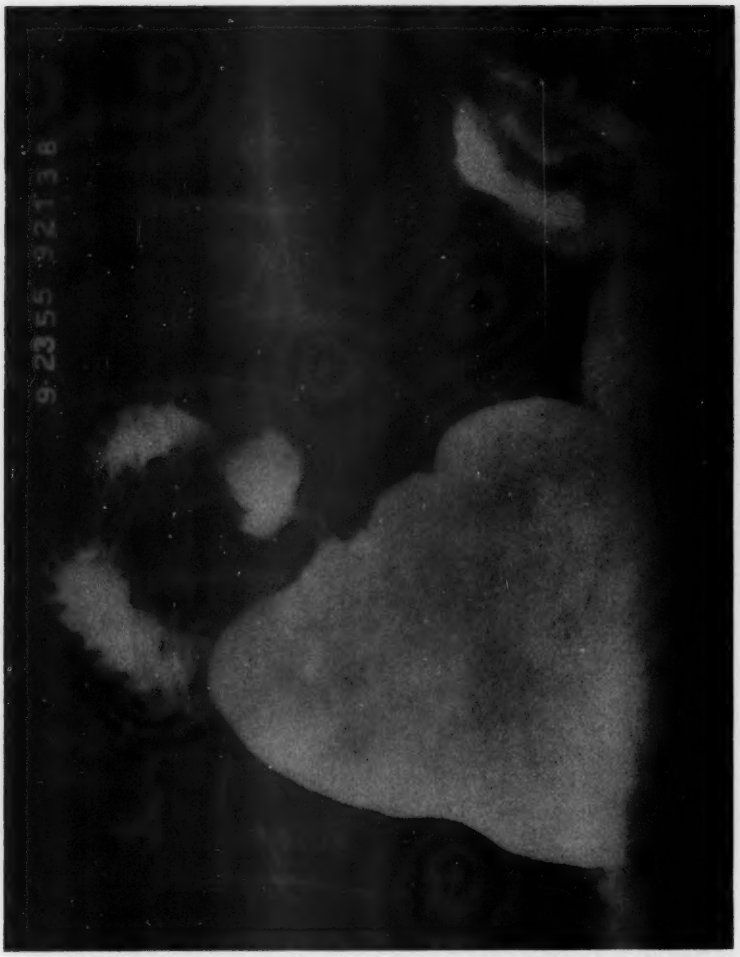

FIG. 4. Giant ulcer in Case 4. Note the pyloric narrowing and adjacent antral deformity which remained constant.

was poor. Vomiting occurred intermittently and slight abdominal distention appeared. A diagnosis of reflex ileus was made. On the ninth postoperative day, the patient complained of generalized abdominal pain more severe in the lower abdomen. There were definite signs of peritonitis and the patient lapsed into shock which only partially responded to blood transfusions. At emergency laparotomy, several liters of small bowel contents were present in the peritoneal cavity. The terminal ileum had perforated from gangrene secondary to an old adhesive band involving the omentum which had obstructed the ileum 10 cm. from the cecum. Several inches of ileum were resected. Postoperatively the patient gradually went into more profound shock and died several hours later. No autopsy was obtained.

Case 5. E. S., a white woman 72 years of age, entered the University Hospital on the medical service on Feb. 16, 1955 complaining of persistent vomiting and epigastric pain.

She had been treated 6 years for recurring epigastric cramping pain by her local physician who had demonstrated gallstones on x-ray examination. There was a history of tarry stools and questionable jaundice. Symptoms had been markedly aggravated over the preceding 2 months. The patient had vomited almost everything ingested and at times the vomitus was coffee ground material. A gradual weight loss from 186 pounds to 98 pounds had occurred during the 6 years of illness and the patient had become addicted to narcotics. On physical examination, the patient was chronically ill, malnourished and too weak to stand. Epigastric tenderness was marked. She was in a severe state of dehydration with alkalosis, hypokalemia and azotemia. A liquid diet and supportive parenteral therapy were instituted. Three days later the patient vomited blood and developed tarry stools. A gastrointestinal series on February 21 although unsatisfactory, suggested an obstruction in the pyloric area, a deformed duodenal bulb and 30 per cent gastric retention after 3 hours. The patient responded poorly to treatment, continued to vomit and maintained a refractory alkalosis and

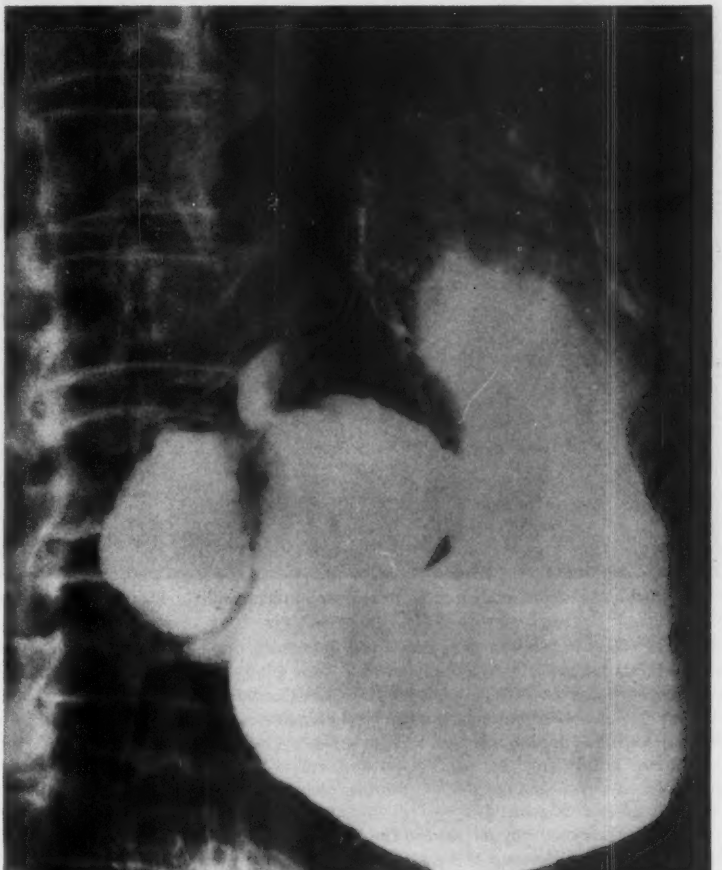

FIG. 5. Large ulcer in Case 5. The barium filling in it was thought to be extra luminal or possibly in the gallbladder in view of later filling of the biliary tree. See figure 6.

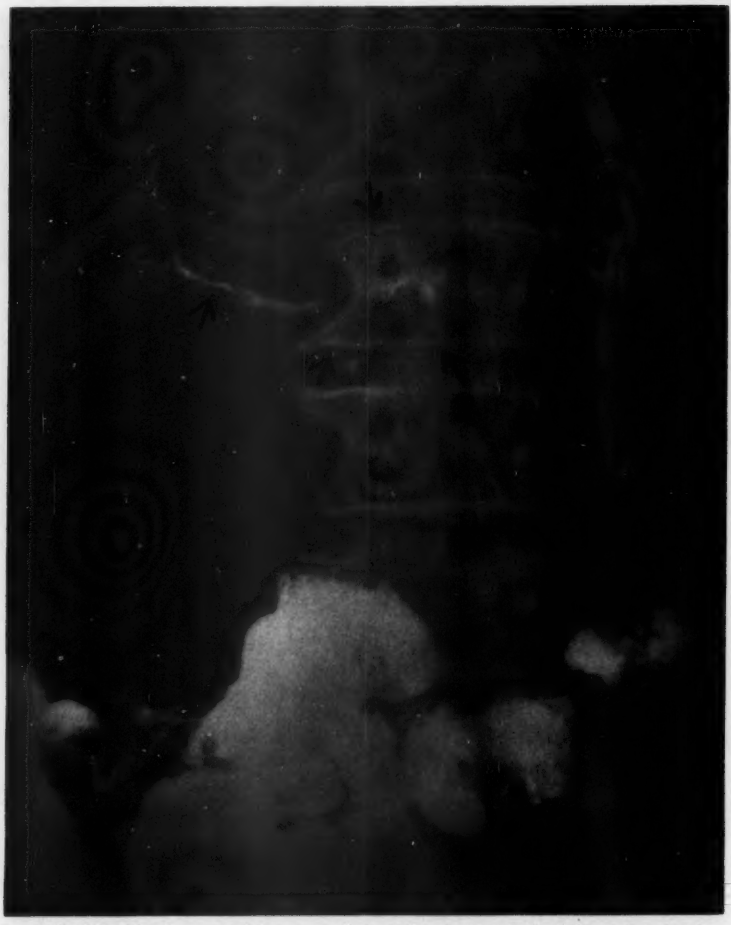

FIG. 6. Residual barium at 24 hours outlining the ulcer and the biliary tree in Case 5

hypokalemia. Gastric suction was employed intermittently. A second gastrointestinal series on March 1 was interpreted as showing some prepyloric deformity of the stomach with a constant large barium collection within the duodenal bulb. Adjacent to this there was an amorphous collection of barium, 3 by 6 cm. considered extraluminal (fig. 5). At 24 hours some barium remained in this area and there was also barium in the common bile duct and lower biliary tree (fig. 6). The patient was transferred to the surgical service but became worse in spite of vigorous supportive therapy in preparation for operation. She died 19 days after admission.

At autopsy a large ulcer was found in the first part of the duodenum. It nearly encircled the lumen leaving only a strip of normal mucosa and contracted wall. A crater 3 by 6 cm. was hollowed out in the pancreas and extended up on the hepatoduodenal ligament into the liver. The rim of anterior duodenal wall was adherent to the eroded liver margin. The common duct had been largely destroyed and opened into the center of the ulcer bed.

FIG. 7. Autopsy specimen Case 5. The arrow is in the eroded common duct in the base of the ulcer.

Scarring just distal to the ulcer nearly obstructed the duodenum (fig. 7). Microscopic examination verified the gross impression of a benign peptic ulcer.

Case 6. E. C., a 72 year old white woman, entered the University Hospital on March 3, 1955 because of persistent vomiting for 3 months. The patient had experienced symptoms suggesting a peptic ulcer for 20 years and had been under medical care elsewhere for this condition for 5 years. There had been three episodes in the past of temporary pyloric obstruction and on occasion coffee ground vomitus and tarry stools had been present. Physical examination disclosed a markedly obese but frail and weak patient with moderate epigastric tenderness. For the next 10 days the patient was treated intermittently with gastric suction and a modified Sippy regime but the pyloric obstruction was never fully relieved. A gastrointestinal series showed a marked duodenal deformity with a double contoured appearance suggesting an ulcer within the bulb and a large diverticulum in the third portion. At 3 hours there was a 10 per cent gastric residual with some barium still remaining in the duodenal bulb and diverticulum (fig. 8, 9).

On March 13 while attempts were continuing to improve the patient's condition for surgical exploration, a sudden hemorrhage precipitated the operation. A giant ulcer 4 by 5 by 1.5 cm. involving the first and second portion of the duodenum was present. This extended into the pancreas to approach the common duct. A single bleeding vessel was present in the ulcer crater and clear fluid, presumed to be pancreatic juice, welled up in the crater after bleeding had been controlled. The gallbladder contained numerous stones. It was necessary to place a metal probe in the common duct during dissection of the duo-

FIG. 8. Ulcer in Case 6 appearing to be simply a deformed duodenal bulb

denal ulcer to identify their relationships. The duodenum was trimmed from the ulcer bed and a Billroth II type gastric resection and cholecystectomy were performed. Satisfactory suture closure of the duodenum was impossible and a catheter duodenostomy was substituted. The ulcer bed in the pancreas was drained. Microscopic sections of the duodenum showed a benign peptic ulcer.

Postoperatively the patient responded well immediately but as time went on after the duodenal catheter was removed, the duodenal fistula showed no tendency to heal and she became a severe nutritional and electrolyte problem. She gradually lost strength and all attempts at vigorous supportive therapy failed. Death occurred on the forty-fourth postoperative day.

At autopsy there were minimal abdominal adhesions. Two fistulous tracts were present, one the common duct still extruding bile and the other extending from the duodenal stump which was still open and showing little tendency to heal. There was no evidence of stomal

FIG. 9. Three hour film in Case 6. The ulcer crater retains barium and is more apparent than on initial film (fig. 8). Note the gastric residual and diverticulum of the third portion of the duodenum.

obstruction at the gastrojejunal anastomosis which could account for this failure to heal. Immediately adjacent there remained an excavated ulcer bed in the pancreas which had contracted to 2.5 cm. in diameter. The main pancreatic duct was lost in the scar tissue in attempting to trace it into the ampulla.

Case 7. E. R., a 77 year old white male diabetic, was admitted to the University Hospital on Feb. 28, 1957 complaining of epigastric pain, anorexia, intermittent vomiting and a loss of 20 pounds during the previous 6 weeks. Several tarry stools had been passed the week before admission. The patient had experienced symptoms compatible with a peptic ulcer for 4 to 5 years. An amputation of the penis for squamous cell carcinoma had been performed in 1951. On physical examination in addition to malnutrition and obvious anemia,

FIG. 10. Ulcer in Case 7. Note the central collection of barium with a surrounding halo and a thin line of barium outlining the periphery of the ulcer.

there was a tender fixed mass approximately 5 cm. in diameter palpable in the epigastrium. Admission hemoglobin was 6.03 grams and hematocrit 23. An admission gastrointestinal series revealed an almost complete pyloric obstruction. A tentative diagnosis was made of carcinoma of the stomach. Under supportive treatment, the patient improved and was able to retain a liquid diet but continued to pass tarry stools. A subsequent gastrointestinal series disclosed a markedly deformed duodenal bulb and postbulbar segment with a large filling defect. The possibility of a giant duodenal ulcer or malignancy was entertained (fig. 10).

At laparotomy there was an inflammatory mass composed of the gallbladder, duodenum, head of the pancreas and greater omentum. Differentiation from a carcinomatous mass was made only after gastrotomy with exploration of the duodenum. A giant ulcer 3.5 by 5 cm. in diameter was encountered on the posterior aspect of the duodenum. The duodenal wall was attached to the entire periphery of the ulcer which had deeply eroded the subjacent pancreas. Distally the ulcer approached close to the papilla of Vater. The gallbladder con-

tained stones. Cholecystectomy with common duct exploration and a Billroth II type of gastric resection were performed. A probe in the common duct through the papilla served as a guide to prevent injury as the duodenum was dissected from the ulcer crater. A modified suture closure of the duodenum was accomplished with difficulty and the stump drained. Microscopic sections of the crater margins of the duodenum disclosed a benign peptic ulcer. A temporary duodenal fistula which closed spontaneously developed postoperatively. The patient was discharged from the hospital asymptomatic on the nineteenth postoperative day.

Case 8. B. B., a 55 year old white woman, was declared incompetent and admitted to the State Hospital for Nervous Diseases in January 1954. A history of "stomach trouble" for which no medical advice had been sought was also obtained from relatives. Five days after admission, the patient developed a picture of an acute surgical condition within the abdomen with free air in the peritoneal cavity. At laparotomy a 1 cm. perforation of the anterior superior border of the first portion of the duodenum was present. This was closed with an omental graft. On the third postoperative day a massive hematemesis occurred which produced shock. The blood pressure and pulse stabilized at normal levels after 1500 cc. of whole blood but several hours later the patient suddenly vomited blood in such copious amounts that she could not completely clear her airway and as a result aspirated a large amount of blood. From this time on she was cyanotic. At emergency laparotomy the previously closed perforation was found to represent only the superior aspect of a large ulcer 5 by 8 cm. in diameter which had left only a strip of normal mucosa on the lesser curvature side and extended circumferentially to almost completely encircle the duodenum. Posteriorly the pancreas along with the main trunk of the pancreaticoduodenal artery had been eroded. After ligation of this bleeding vessel, a Billroth II type gastric resection was performed. It was possible by tedious dissection to free sufficient posterior duodenal wall to close the duodenal stump. Bronchoscopy after hemorrhage was controlled showed the tracheobronchial tree to contain a large quantity of blood clots extending into the smallest radicals visualized. After strenuous efforts to suction this material from the bronchi, the patient still remained cyanotic and died several hours after returning to the ward. An autopsy was not obtained. Microscopic sections of the ulcer margin disclosed a benign peptic ulcer.

DISCUSSION

Clinical Manifestations: While a giant ulcer of the duodenum presents no symptoms unique from those of other peptic ulcers and may, in fact, mimic symptoms indicating pathology of other organs in this area, there are particular features which should arouse suspicion. All patients except 1 were over 40 years of age and all except 1 gave a history of long standing ulcer symptoms which responded poorly to medical management. Epigastric pain often is more intense than in the usual ulcer patient and may be referred more to the right upper quadrant or posteriorly into the back. One patient became a morphine addict. Perhaps the fact that the patients were uncooperative or some unable to cooperate, i.e. psychotic, chronic alcoholic, drug addict, accounts for the poor medical response and subsequent development of a giant sized ulcer.

Massive hemorrhage or pyloric obstruction or both are most likely to be the presenting problem as a climax to a period of exacerbation. If not, one or both complications are almost certain to have occurred in the past. Perforation into the free peritoneal cavity in Case 8 of this series is the only such reported instance of this complication. Because of anatomic location, perforation usually occurs as a slow penetration so that adjacent structures or organs wall off the area of destroyed duodenal wall.

Roentgenographic Findings: Giant ulcers are frequently missed entirely because they are so large the ulcer crater is interpreted as being a normal or slightly deformed bulb. As pointed out by Brdiczka however, the contours of the crater are smooth and the walls rigid with a lack of the mucosal pattern or radiating rugae seen in usual duodenal ulcers. He further described a narrowing from spasm or organic constriction of the duodenum just distal to the ulcer which may be present and interpreted as a stricture distal to a normal bulb (fig. 3). In other instances an obviously abnormal contour of the bulb may be considered simply a deformity rather than a giant ulcer because the size of the deformity is believed to be too large to be an ulcer crater (fig. 8). If present a smaller fleck of increased density of barium within the deformed area may be interpreted as the active ulcer. This latter may represent the most deeply penetrated niche in the pancreas (fig. 10). Reflex spasm or organic occlusion at the pyloric area may preclude enough barium from entering the duodenum for accurate examination and simulate an antral lesion (fig. 4). A collection of barium may be considered as extra luminal, a diverticulum or pseudodiverticulum; however, no stalk can be demonstrated (fig. 5). The ulcer crater has a tendency to stand out more at times as the duodenum empties its barium or it may retain an air bubble (fig. 9). Figure 6 illustrates such an instance where communication with the common duct was also demonstrated on the 24 hour film which showed the biliary passages outlined with barium.

It is quite apparent that these many variations make the radiologic diagnosis difficult and a high degree of suspicion is required on the part of the radiologist to arrive at the correct diagnosis. Sometimes no abnormality can be recognized on close scrutiny of x-ray films after an exact diagnosis is made at surgery or autopsy.[9]

Diagnosis and Treatment: While the presence of a giant ulcer of the duodenum may be completely obscure and defy accurate preoperative diagnosis, the majority of patients have clinical symptoms suggesting an ulcer and roentgenologic evidence of a pyloric or duodenal abnormality. In some instances, these findings will allow the diagnosis to be entertained with a high degree of certainty but in the majority of patients surgical exploration will be necessary. In this series the correct diagnosis was suspicioned from roentgenograms in 2 patients. In 4 patients the diagnosis was made at surgical exploration and in 2 instances the condition was recognized only at autopsy. The need for an aggressive surgical attitude is apparent in dealing with the problem of hemorrhage or obstruction in patients with a history of long standing chronic ulcer symptoms. Occasionally this will demand a recommendation for exploration in the face of what for all intents and purposes is a normal roentgenologic picture of the stomach and duodenum. If the roentgenologic findings suggest the possibility that such a lesion is present, surgery becomes mandatory at the earliest possible moment. It seems exceedingly doubtful that an ulcer over 2.5 cm. could be expected to heal satisfactorily under medical management and the hazard of death from hemorrhage during treatment is so great as to preclude any attempts along this line.

In retrospect, it is quite evident that most of the patients in this series should have been considered for definitive surgical procedures at an earlier period in their disease as well as handled more aggressively after their hospitalization.

At surgical exploration the diagnosis may be initially as confusing as preoperatively. A presenting mass may be confused with a neoplasm of the pancreas, duodenum or stomach. Extensive inflammatory adhesions and involvement of adjacent structures make dissection hazardous. Early in the procedure a direct approach through the duodenal mass or distal stomach may be necessary to establish the diagnosis or to control a continuing massive hemorrhage arising from the pancreaticoduodenal artery. A frozen section may be required to exclude malignancy. In Bullock and Snyders patient, the true situation was recognized only after a mass including the duodenum and part of the pancreas and colon had been resected under the impression that a carcinoma was present.

A gastric resection with removal of the involved duodenum is recommended. The danger of continuing or recurrent hemorrhage following an exclusion procedure or vagotomy and gastroenterostomy in addition to the unlikeliness of satisfactory healing of the large ulcer condemns such procedures if they can possibly be avoided. The extensive involvement and destruction of the duodenum eliminate a Billroth I operation, in fact simple suture closure of the duodenal stump may be exceedingly difficult or impossible. Some alternative procedure such as catheter duodenostomy or approximation of the anterior duodenal wall to the distal ulcer margin may become necessary. A probe placed in the common duct and through the papilla of Vater is very helpful in preventing injury to these structures. An almost insurmountable problem exists if either or both the common and pancreatic ducts enter in the ulcer crater in a severely ill, nutritionally depleted, and perhaps bleeding patient. Here an exclusion procedure or a two stage gastric resection after the method of McKittrick may be a necessary compromise after initially controlling the bleeding vessel.

Analysis of Mortality: Three of 8 patients survived and are apparently well. One patient died within a few minutes from massive bleeding before therapy could be started. Copious hematemesis in 2 patients led to the aspiration of blood which was believed to be the instigating cause of death in one. If bronchoscopy with successful aspiration of the bronchial tree is carried out early, dramatic improvement may rapidly occur and the patient be converted from a poor operative risk into a more favorable one. One completely avoidable death occurred as a result of confusing an acute mechanical small bowel obstruction with a postoperative reflex ileus until a terminal phase had been reached. In one patient continued secretion of bile from a common duct fistula and duodenal and pancreatic secretions from a catheter duodenostomy played a part along with malnutrition and general debility in causing eventual death. An exclusion procedure or two stage gastric resection might have circumvented this fatality provided no further hemorrhage would have occurred following suture of the active bleeding point in the ulcer. A similar policy might have averted the death of one patient who never could be adequately prepared for laparotomy because

of malnutrition, general debility, and refractory hypokalemic alkalosis. It is extremely doubtful however that this latter patient could have withstood any definitive surgical procedure during the period of her hospitalization.

The management of patients with these large ulcers will undoubtedly continue to remain unsatisfactory because of difficulties in diagnosis and treatment. Only 7 patients of the 21 whose cases have been reported including this series survived, giving a mortality rate of 62 per cent. It should be emphasized that earlier definitive surgical therapy in the duodenal ulcer patient who responds poorly to medical management or fails to cooperate may circumvent the ultimate development of a giant ulcer with its inherent greater morbidity and mortality.

SUMMARY

Eight patients with giant benign duodenal ulcer are reported with 3 patients surviving. In only 2 instances was such a lesion suspected before operation or autopsy.

A giant ulcer may be present in the duodenum when roentgenologic studies indicate a normal or only slightly abnormal appearing duodenal bulb.

Diagnosis is difficult. Certain clinical features and roentgenographic changes may arouse suspicion but surgical exploration is usually necessary for an accurate diagnosis.

The condition appears uniformly fatal from hemorrhage or its complications unless surgical intervention is undertaken.

Surgical treatment carries an unduly high morbidity and mortality rate.

Giant duodenal ulcers are believed to exist more frequently than the accumulated literature suggests.

Earlier surgical intervention is urged in the uncooperative or medically resistant chronic ulcer patient especially if bleeding has been a symptom.

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THE DUMPING SYNDROME AND ITS SURGICAL TREATMENT

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The phenomenon of "dumping" can be precipitated in any individual by a sufficient quantity of a concentrated solution distributed into a sufficiently large portion of small bowel. The term "dumping" probably originated from the original concept that this phenomenon was caused by introducing a large amount of food into the small bowel rapidly. Somewhat later, Machella[1] reported interesting studies on this condition, concluding that the phenomenon was the result of excessively stretching the serosa of the jejunum particularly, not merely by the food introduced but primarily because of the bulk of fluid pulled into the bowel from the extracellular space by the hypermolar contents of the bowel. More recently, Randall,[2, 3] and his associates, restudied this problem and demonstrated nicely some of the physiologic abnormalities resulting from the transfer of fluid into the lumen of the bowel. The symptoms were not due primarily to distention of the bowel but rather to depletion of the extracellular fluid. They demonstrated alterations in circulating blood volume, electrocardiographic changes reminiscent of ischemia of cardiac muscle and reduction of cardiac output. Subsequently, studies have been performed in which there was some question about the profound changes in circulating blood volume in all cases of "dumping". It might well be that some of the symptoms result from intravascular pooling of the blood rather than actual transfer of fluid to the lumen of the gastrointestinal tract.

We can agree that "dumping" occurs when a relatively large amount of a hypertonic solution is distributed throughout a large portion of the small bowel. There are differences of opinion as to the definitive physiologic alterations. My studies of patients with the more pronounced symptoms reveal barium-marked food entering the small bowel abruptly, and being rapidly propagated along its entire length, reaching the illeocecal valve usually within 20 minutes from the time of ingestion. Apparently this distribution of material through long segments of bowel is an important factor in the precipitation of the characteristic symptoms.

The phenomenon of "dumping" can and does occur following all types of gastric surgery. Careful questioning of patients who have undergone gastric surgery will demonstrate this phenomenon occurs in approximately 50 per cent. Fortunately, the symptoms are usually mild and patients accept this inconvenience because they are otherwise greatly improved. The symptoms become less severe with time in about 15 per cent. Most of the remaining patients learn to live with the condition by dietary regulation. Unfortunately, these self-imposed

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restrictions are not completely corrective and may occasionally augment the condition. The patient may have been advised to restrict fluids at mealtime. The patient not only restricts fluids then, but the entire fluid intake is reduced to the point of dehydration and a shrunken blood volume. Then follows loss of appetite, loss of weight, weakness, debility and a greater susceptibility to extracellular fluid shifts. The condition becomes something of a vicious cycle.

These patients can usually be controlled by careful medical management. This regimen must include correction of the shrunken blood volume, adequate hydration of the patient and the consumption of sufficient food to restore the patient to near normal weight. Dehydration is corrected and hemoglobin determined. Any relative anemia is fully treated by transfusion. Simultaneously one of the parasympatholytic drugs is given to the point where there are definite side effects including an increase in pulse rate. Drugs such as Pamine and Banthine given in subtoxic doses reduce the motility of the bowel and lessen secretion. Pancreatic secretion can be practically stopped under these conditions. The secretion of bile is not affected. The decrease of pancreatic secretion is so pronounced that spontaneous closure of acute pancreatic fistulas can be expected routinely. Forcing fluids between meals and taking a minimum of 250 ml. approximately 15 to 20 minutes before a meal is of great importance. Forcing fluids is of sufficient importance to warrant every effort made to find the fluids the patient will take and can tolerate. Zollinger has suggested the use of beer. This beverage has several desirable properties. The salt content is low and the caloric value is considerable. The alcohol adds not only to the caloric intake but also to the patient's feeling of well-being. The bitter taste may stimulate the appetite. The carbohydrate foods cause the symptoms most readily because they are either low molecular weight sugars or starches which undergo rapid digestion, the digestion beginning in the mouth where ptyalin is admixed during chewing. Consequently, the carbohydrates are promptly eliminated from the diet. This elimination leads to poor food intake because a diet without carbohydrates is not usually relished. They should be included in the diet. However, it is necessary to prevent rapid digestion which can be accomplished by impregnating the carbohydrates with fat. Only the crude carbohydrates such as starches are incorporated in the diet in large quantities. The fat prevents contact between the starches and the water soluble digestive ferments. Toast fried in butter, dry cereals impregnated with fats, potato chips, french fried and shoestring potatoes, crackers such as Ritz containing a large amount of fat, various crisp cookies containing large amounts of shortening, sweetened with sucaryl or saccharin rather than sugar, fried rice, hard carbohydrate preparations such as pretzels, doughnuts sweetened with saccharin, etc. are well-tolerated. These fats supply many needed calories. Ordinarily, the meal is taken in relatively dry form. However, if "dumping" should occur, the patient should be urged to drink immediately 250 to 500 cc. of water, tea, coffee, beer or some other fluid which does not contain electrolytes or sugars.

There are individuals, however, who fail to respond to this treatment or who cannot tolerate the diet. Also, another group of patients, those who have had

either subtotal or total gastrectomy, may well not respond to this regimen. A satisfactory pouch for replacement of the stomach has not been described. They all empty very rapidly, with the possible exception of the pouch made from the jejunum.

SURGICAL APPROACH TO THE TREATMENT AND CONTROL OF "DUMPING"

If our conception that "dumping" is due to the abrupt introduction of large quantities of food into the small bowel is correct, then a reservoir which would permit the ingestion of a large amount of food at a meal, but at the same time would act as a true reservoir and empty slowly and piecemeal, should prevent this phenomenon. The surgical attack is directed towards this end. In general a small stoma is presumed to slow down emptying, and a gastroduodenostomy is expected to empty more slowly than a gastrojejunostomy. Therefore a small stoma is constructed and a Bilroth I is perferred to a Hofmeister procedure. "Dumping" may or may not occur in any of these circumstances and converting from one arrangement to the other or vice versa may or may not improve the situation. It is all quite indefinite and certainly most unsatisfactory. A reservoir

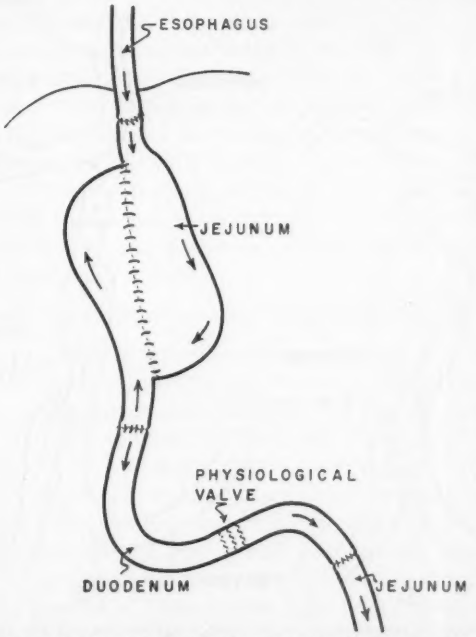

FIG. 1. An illustration of the basic principles involved in the construction of a pouch incorporating an outlet with antiperistalsis for gastric substitution. The entire pouch consists of a single loop of jejunum 20 inches long with its mesentery intact. The isoperistaltic segment attached to the esophagus prevents regurgitation and esophagitis. The antiperistaltic outlet, the length of which fortunately is not critical, anastomosed to the duodenum retards the passage of food and gastric emptying. Emptying occurs piecemeal and requires from 3 to 6 hours for passage of the major portion of the contents of the pouch.

of sufficient volume can be made from a number of different intraabdominal structures such as segments of the large bowel or jejunum, but to obtain one which will retain food and empty piecemeal represents a more difficult problem. The introduction of a segment of bowel with reversed peristalsis at the outlet might be the answer. A method for accomplishing this procedure without incorporating the hazard of compromised blood supply to a short segment of bowel with reversed peristalsis has been realized and is illustrated in figure 1. A pouch made from jejunum with an end to side esophagojejunostomy permits regurgitation into the esophagus and esophagitis results. It therefore will be necessary to incorporate an isoperistaltic segment of bowel between the esophagus and the substitution pouch especially when the outlet of the pouch consists of an antiperistaltic segment of jejunum. The illustrations indicate how this is accom-

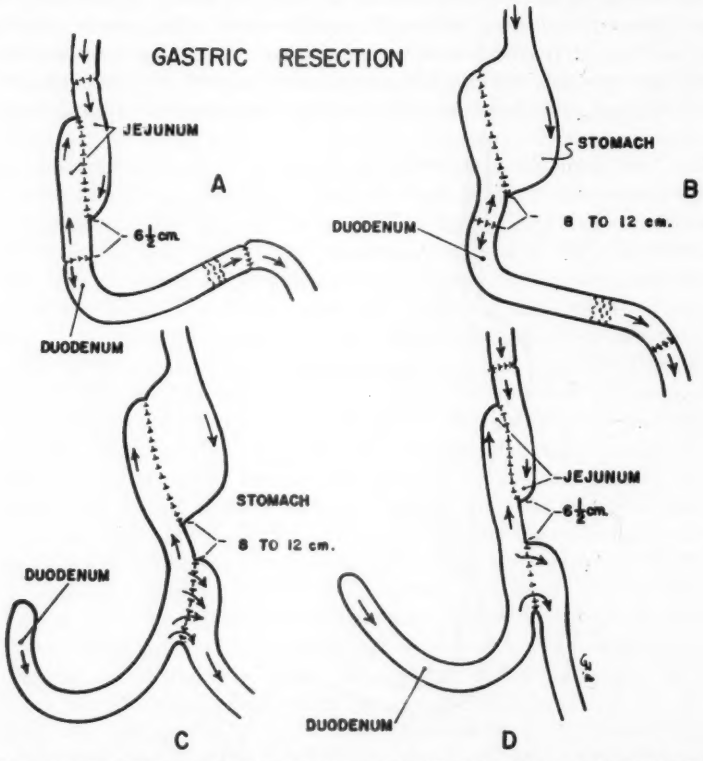

FIG. 2. A diagramatic representation of four modifications of the technical principles presented in figure 1. *A* and *B* demonstrates the arrangement whenever the duodenum is available and suitable for use. *C* and *D* demonstrates the arrangement whenever the duodenum is either not available or unsuitable for use and is excluded. *A* and *D* demonstrates the technic following total gastrectomy. *B* and *C* demonstrates the technic following subtotal (90 to 95%) gastrectomy. Technic *A* has been used in 2 patients. Technic *B* has been used in 1 patient. Technic *C* has been used in 2 patients.

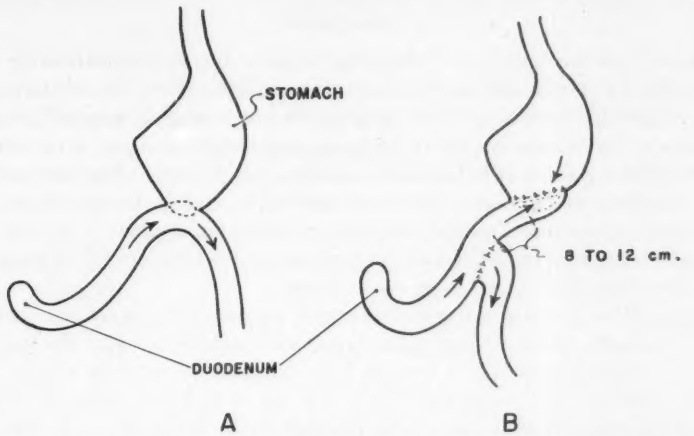

FIG. 3. A schematic illustration of the conversion of a Hofmeister type resection to a modification of technic demonstrated in figure 2 C. This procedure has been used in one instance to convert a Polya type resection to modification illustrated in this figure 3 B.

plished without embarrassment of the blood supply and without the introduction of circular anastomoses at both ends of the short antiperistaltic bowel segment.

The principle of construction of the antiperistaltic outlet in various types of gastric pouches is presented schematically in figure 2. Figure 3 demonstrates a maneuver by which the ordinary Hofmeister gastric resection can be converted into a pouch of increased size with the introduction of the principle of the reversed peristaltic outlet. Fortunately, the length of this antiperistaltic segment is not critical. When the gastric pouch includes a remnant of the stomach with its strong musculature, the segment with reversed peristalsis should be longer than when jejunum alone is utilized.

POSTOPERATIVE OBSERVATION OF GASTRIC SUBSTITUTION POUCHES

These pouches have been observed as long as a year. A meal designed to cause "dumping" has been administered without the occurrence of the phenomenon in a single instance. Even the administration of 500 gm. of glucose dissolved in grape juice has failed to precipitate the syndrome. Food mixed with barium has been given and the manner and rate of gastric emptying observed by fluoroscopy. Emptying occurs in the course of 3 to 6 hours in a piecemeal manner. Relatively small quantities of material are delivered to the small bowel in a progressive manner. Small traces of barium may remain in the pouch after 12 hours. This small amount of residual material does not interfere with appetite.

Admittedly, the number of patients who have been treated is too small and the period of observation is too short to permit dogmatic conclusions, but the results are promising. The patients eat well and have either gained weight or maintained their normal weight. There has been no instance of "dumping" either in the course of normal eating or in a determined effort to precipitate the phenomenon by the ingestion of large quantities of carbohydrates.

DISCUSSION

Usually the phenomenon of "dumping" when it occurs postoperatively can be controlled by proper diet and medical management. There are instances, however, where this procedure is not satisfactory and a surgical approach would be welcomed. The increasing use of subtotal gastrectomy in cases of carcinoma in the prepyloric portion of the stomach and total gastrectomy where the carcinoma is in the body of the stomach or near the cardia makes the construction of a functional substitution gastric reservoir of added importance. A technic incorporating a segment of bowel with reversed peristalsis is described. Regurgitation into the esophagus has not been encountered.

What will be the fate of the antiperistaltic segment? Will it remain unchanged in its character, will it become dilated, perhaps markedly so with the loss of any valvelike function, or will it become hypertrophied even to the degree that it might interfere with emptying? While it is too early to be dogmatic about the fate of the segment of antiperistaltic jejunum at the outlet, it can be stated that to date observation by fluoroscopy does not show the segment to have become dilated and functionless, but rather it has become somewhat hypertrophied. The period of emptying this substitution pouch has not become shorter with the passage of time, but has either remained essentially the same as it is 3 weeks after operation or has become slightly longer.

These preliminary observations would indicate that a satisfactory substitution pouch might well have been constructed. The procedure appears worthy of further trial and observation.

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THE SIGNIFICANCE OF THE ANOMALOUS ORIGIN OF THE LEFT HEPATIC ARTERY FROM THE LEFT GASTRIC ARTERY IN OPERATIONS UPON THE STOMACH AND ESOPHAGUS*

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Every surgeon is cognizant of the fact that anomalous anatomic variations in the areas of "every-day operations" are to be found, but the significance of some of the abnormalities seen at operation may not be clear. Occasionally some morbid or fatal complication may occur as a result of interference, knowingly or unknowingly, with a congenital deviation from the usual "normal" anatomy. The significance of one of the many anomalies of the arterial supply to the stomach and liver, occurring frequently enough to warrant reemphasis, is described here.

The left lobe of the liver occasionally receives its arterial blood supply through a branch of the left gastric artery. This branch may be the sole source of arterial blood to the left lobe of the liver (anomalous replaced left hepatic artery) (fig. 1), or it may be an accessory branch (in addition to a normally situated but small left hepatic artery) (fig. 2). Division and ligation of such an anomalous left hepatic artery (replaced left hepatic artery) can result in fulminating necrosis of the left lobe of the liver and death of the patient, as did occur in one patient and which will be described.

Normally the celiac axis supplies the upper abdominal viscera through the splenic, left gastric and common hepatic arteries. Typically the hepatic artery supplies three branches to the liver: the right, the left, and the middle hepatics. The last furnishes the arterial blood supply to the quadrate lobe (medial segment of the left lobe). When the right or the left hepatic does not arise normally from the celiac hepatic artery, the missing right or left hepatic is replaced from another source (the right hepatic, prevailingly, from the superior mesenteric; the left hepatic from the left gastric). Such replaced hepatics are not to be confused with accessory hepatic arteries which are additive to the normally present right and left hepatics derived from the celiac. Michels, in his detailed and complete descriptions of the vascular anatomy obtained from anatomic dissections of 200 bodies, found that an aberrant left hepatic artery arises from the left gastric artery in 46 out of 200 dissections (23 per cent)[5]. He revealed, furthermore, that of these 46 patients with aberrant left hepatics from the left gastric, one-half (11.5 per cent of total number) were replaced left hepatics (the only blood supply to the left lobe of the liver) and the other half were accessory arteries to the left lobe. The accessory hepatic arteries, even though accompanied by normally pres-

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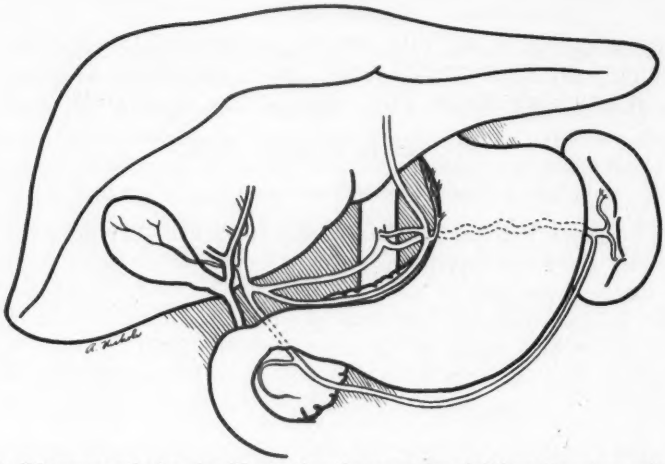

FIG. 1. Diagrammatic drawing illustrating the anomalous left hepatic artery arising from the left gastric artery and supplying the left lobe of the liver. Since there is only the right hepatic artery from the hepatic proper, the anomalous vessel is a replaced left hepatic artery. Note the acute angle formed at its origin from the left gastric artery.

ent hepatic arteries, are considered by Michels to be nonexpendable, based on the work of Healey and Schroy,[4] who studied 70 plastic casts of excised autopsy specimens of the liver. The latter studies indicate that "functionally considered, there are no accessory hepatic arteries and that each hepatic artery, whatever its source, has a selective distribution to a specific area of the liver parenchyma." In a roentgenographic study of the arterial supply of the liver, Glauser[3] also demonstrated that each hepatic branch has a selective distribution in the liver. Because of the frequent origin of a left hepatic (replaced or accessory) from the left gastric artery, the latter artery was designated as the "gastrohepatic artery" by Haller and early anatomists[5]. Adachi found that the left gastric artery contributed to the blood supply of the left lobe of the liver in 20 per cent of instances.[2] Anson states that variations in the hepatic anatomy are common and that "not uncommonly two large hepatic arteries are present, the left being a derivative of the left gastric . . . ."[2]

When an aberrant left hepatic arises from the left gastric artery it is given off by the latter at a rather acute angle. The aberrant left hepatic artery courses superiorly and to the right between the two layers of the lesser omentum, crosses the caudate lobe and enters the liver via the fissure for the ligamentum venosum. The pulsatile artery may vary in size from 2 to 5 mm. in diameter. When the structure does not pulsate, it is likely that a rudimentary ligament only is present in this location.

The knowledge of the anomalous blood supply to the liver becomes of practical importance in operations on the stomach and esophagus. In operations on the gallbladder, such as cholecystectomy, the cystic artery is usually ligated as close to the gallbladder as is feasible; utilizing this technic, one is not likely to injure

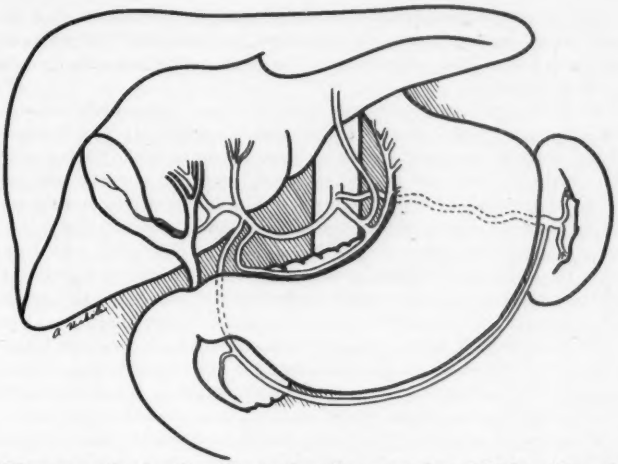

FIG. 2. Diagrammatic drawing illustrating the anomalous left hepatic artery arising from the left gastric artery and partially supplying the left lobe of the liver. The aberrant vessel is designated as an accessory left hepatic artery because a normally situated left hepatic artery may also partially supply the left lobe.

the hepatic arteries even when there is an anomalous position of the cystic artery. On the other hand, in operations for cancer of the esophagus and stomach in which extensive regional lymph node excision is practiced, the site of ligation of the left gastric artery may be of utmost consideration. If it is found that the left gastric artery contributes to the arterial supply of the left lobe of the liver by way of a pulsating aberrant left hepatic artery (particularly a replaced left hepatic artery, in which case there is no left hepatic artery from the proper hepatic artery), then the left gastric artery must be ligated distal to the take-off of the left hepatic artery. The lymph nodes in this instance must be dissected away from these vessels, a task more delicate than the usual ligation of the left gastric artery at its origin from the celiac axis with accompanying lymph node dissection. In gastric resections for peptic ulcer or esophageal operations for benign diseases, similar considerations should be taken in the selection of the site of ligation of the left gastric artery distal to the aberrant left hepatic artery, although there are no compelling reasons for proximal ligation in benign disease.

The significance of the anomalous origin of the left hepatic artery from the left gastric artery is exemplified in the following case reports.

CASE REPORTS

J. S., a 58 year old Negro man, was admitted to the hospital on April 23, 1954. There was a history of progressive dysphagia of 4 months duration, anorexia, and a weight loss of 20 pounds since the onset of symptoms. His health was good prior to the onset of symptoms. Physical examination revealed no abnormalities except evidence of weight loss and a blood pressure of 190 systolic, 100 diastolic. The blood count was within normal limits and urinalysis indicated a specific gravity of the urine of 1.010 and a trace of albumin. The nonprotein-nitrogen was 49 mg. per cent, the total proteins were 6.6 gm. per cent, with normal

fractional ratio, the electrocardiogram was normal, and the roentgen film of the chest was within normal limits. Barium studies demonstrated an obstructive, irregular defect in the thoracic esophagus at the level of the arch of the aorta, consistent with the appearance of a carcinoma of the esophagus.

On April 26, 1954, a palliative esophagectomy was done under endotracheal anesthesia through both an upper abdominal and a right thoracic approach. The abdominal portion of the operation, through an upper midline incision, consisted of the freeing up of the upper stomach with ligation of the left gastric artery at its origin and the left gastroepiploic vessels, and the appropriate lymph node dissection. (No anomalous vessels were noted or recorded). After surgical closure of the abdominal incision, a right transthoracic exploration was made through the periostial bed of the resected right sixth rib. A carcinoma at the junction of the upper and middle thoracic esophagus, extending superior to the azygos vein and adherent to the posterior surface of the trachea, was found and excised with the entire distal esophagus and the mediastinal lymphatics. The stomach was drawn through the hiatus and anastomosed to the upper esophagus in two layers with interrupted silk sutures, after suture closure of the cardial opening of the stomach. This was considered a palliative resection because of the tumor attachment to the membranous trachea.

During the thoracic portion of the operation there was a transient period of hypotension (80/60) which responded to added intravenous infusions of whole blood. The lesion was an ulcerating squamous cell carcinoma of the esophagus with extension to the posterior trachea with no metastases to the lymph nodes.

Postoperatively there were recurrent episodes of hypotension with the development of oliguria, and an increase in secretions within the tracheobronchial tree. There was increasing restlessness, perspiration, some lower abdominal pain with temperature elevations to 101° and 100° rectally, pulse 88 to 110 per minute, and systolic blood pressure fluctuations from 120 to 90 mm. Hg. The patient died during the second postoperative day in shock in spite of treatment with whole blood, intravenous fluids, antibiotic drugs, tracheal suctioning, and other supportive measures. There was an elevation of the nonprotein-nitrogen to 116 mg. per cent on the day of death.

Postmortem examination revealed acute and recent infarction of the left lobe of the liver, questionable infarction of the fundus of the stomach, a ligated aberrant left hepatic artery, an intact esophagogastric anastomosis without tumor, residual carcinoma on the posterior surface of the trachea, and bilateral chronic pyelonephritis. Histologic section of the left lobe of the liver demonstrated "infarction with marked necrosis of the central areas of the lobules and only a few living cells around the portal areas . . . . In other parts of the section the entire lobules are undergoing necrosis with breaking up of cells and infiltration of phagocytes is taking place." These changes were described as having an appearance of resulting from occlusion of the arterial, not the portal, supply to the liver. There was no evidence of embolic occlusion of hepatic arterial branches.

Comment: Death of the patient on the second postoperative day following palliative esophagectomy for carcinoma of the esophagus is considered in this instance to be due to infarction of the left lobe of the liver owing to ligation of a replaced left hepatic artery originating from the ligated left gastric artery. The chronic pyelonephritis was probably an abetting condition.

C. L., a 61 year old white man, was admitted to the hospital on Dec. 12, 1954, with a history of epigastric pain and anemia due to upper gastrointestinal hemorrhage. Roentgen examination revealed a defect in the body of the stomach consistent with neoplasm. After preoperative preparation of the patient, a malignant lymphoma was excised by subtotal gastrectomy, during which operation an anomalous left hepatic artery was visualized originating from the left gastric artery. The accessory left hepatic artery was preserved by ligation of the left gastric artery distal to the aberrant vessel. Postoperative recovery was uncomplicated, during which time radiation therapy was given.

J. B., a 57 year old white man, was admitted to the hospital on March 4, 1955, with a history of epigastric pain and repeated vomiting. Roentgen examination demonstrated an

ulcer on the lesser curvature of the stomach. At operation a subtotal gastric resection for lymphosarcoma of the stomach and a cholecystectomy for cholelithiasis was done. During the operation an anomalous pulsating left hepatic artery arising from the left gastric artery was observed and preserved by ligation of the left gastric artery distal to the accessory hepatic artery. Postoperative recovery of the patient was uneventful.

A. J., a 62 year old white woman, was admitted to the hospital on Oct. 10, 1955, with a 2 year history of pain, nausea, vomiting, and recent hematemesis. Roentgen examination demonstrated an obstructing ulcer in the prepyloric area. At operation an obstructing duodenal ulcer was found, for which a subtotal gastric resection was done. During the operation an anomalous hepatic artery was visualized and palpated, arising from the left gastric artery. Dissection of the porta hepatis as well as the hepatic branches of the celiac axis revealed a right and a left hepatic artery from the proper hepatic artery. The left hepatic branch was much smaller in caliber than is usual, however. The accessory left hepatic artery was preserved by ligation of the left gastric artery distal to the anomalous origin of the left hepatic artery. There were no complications postoperatively.

E. H., a 47 year old Negro man, was admitted to the hospital on Jan. 6, 1956, with a history of abdominal pain and recent hematemesis. Roentgen studies demonstrated an obstructing duodenal ulcer. At operation a subtotal gastric resection was done for two large obstructing, penetrating duodenal ulcers. During operation an anomalous (replaced) left hepatic artery was noted to arise from the left gastric artery and was preserved by distal ligation of the left gastric artery. There were no complications postoperatively.

C. G., a 64 year old white man, was admitted to the hospital on March 10, 1957, with a history of 7 years duration consisting of recurrent epigastric pain, occasional tarry stools and a 20 pound weight loss. Roentgen studies demonstrated a duodenal ulcer, a small sliding hiatal hernia, and a nonfunctioning gallbladder. A transabdominal operation was done at which time a penetrating duodenal ulcer with inflammatory reaction about the porta hepatis and cystic duct was found, as well as the small hiatal hernia. A subtotal gastrectomy was carried out. During operation an anomalous (replaced) left hepatic artery arising from the left gastric artery was found and preserved by ligation of the left gastric artery distal to the origin of the aberrant left hepatic artery. The postoperative course was uncomplicated.

The incidence of anomalous left hepatic artery (either replaced or accessory) arising from the left gastric artery in the personal experience presented here (6 out of approximately 100 patients operated upon for esophageal and gastric diseases in 3 years) is not as high as that reported by anatomists (20 to 23 per cent). It is possible that further cases are overlooked, but it seems more likely that nonpulsatile ligamentous structures which have also been seen in the lesser omentum during operations in the last 3 years may not be functioning arteries. This nonpulsatile structure has been noted more often than the pulsating vessels described in the reported cases above, which may account for the discrepancy in frequency. Another disparity in incidence between reported anatomic reports and clinical experience, generally, becomes apparent when it is considered that many surgeons divide and ligate the left gastric artery at its origin routinely with lymph node dissection when doing gastrectomies or esophagogastrectomies for carcinoma.

SUMMARY

It is suggested that the surgeon, when operating for gastric or esophageal disease, should search for an anomalous left hepatic artery arising from the left gastric artery. This aberrant structure should not only be visualized but pal-

pated gently for the presence or absence of pulsation. If it is pulsatile, one should avoid interfering with it for fear of endangering the blood supply to the left lobe of the liver. Preservation of vascular integrity to the liver in such instance can be assured by the ligation of the left gastric artery distal to the origin of the aberrant artery, whether it be a replaced or an accessory left hepatic artery. Since the reported anatomic incidence of anomalous left hepatic artery arising from the left gastric artery is stated to be about 1 in 4 in the population, certain precautions should be taken. A replaced left hepatic artery apparently occurs in 1 out of 8 or 9 cases and is the sole blood supply to the left lobe of the liver. An accessory left hepatic artery arising from the left gastric artery occurs in equal frequency. Although the latter is designated as an accessory vessel, there is anatomic evidence of its selective distribution to the liver, which requires that the accessory vessel be preserved as well.

The present report describes 6 instances of anomalous left hepatic artery (replaced and accessory hepatic) arising from the left gastric artery in patients operated upon for gastric and esophageal disease. In the first patient this vessel was unknowingly divided and ligated, which resulted in fatal infarction and necrosis of the left lobe of the liver. In the remaining 5 patients this vessel was recognized and preserved, leading to uncomplicated postoperative courses in these patients.

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AN OPERATIVE PROCEDURE FOR PILONIDAL CYST AND SINUS

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A pilonidal cyst and sinus is an epithelial lined pocket which may have one or more sinuses. It is of congenital origin, located in the midline of the sacrococcygeal region and superficial to these structures. It represents remnants of the obliterated lower portion of the neural canal or an invagination of the surface epithelium. From a study of human embryos, structures forming the sinus are derived by a process of ectodermal invagination from the skin, appendages, hair follicles and sebaceous glands during the embryos third and fourth months of life. Although it is congenital, trauma may play a part. The defect manifests itself around the second and third decade in life.

Pathology: A postanal dimple observed in infants is the simplest form. One or more sinuses may be present lined with epithelium containing hair follicles or sweat glands extending upward, downward, or laterally from 1 to several centimeters. The sinuses are usually located in the midline, and hair may protrude from the sinus openings to one or the other side of the midline. They are not infrequently present as a result of a burrowing infection. The presence of cysts or cavities recorded is 25 per cent while the presence of hair in cysts or sinuses is 40 per cent. The condition is most frequently located between the skin and the sacrum and coccyx usually where the neurenteric canal opens on to the skin. Some may even connect with the neural canal and be the rare cause of meningitis. The patient is usually not aware of this condition until it is brought to his attention by infection. It then may assume considerable proportions and present stages of inflammation, redness, tenderness and swelling. It may be acute with abscess formation or chronic in which the epithelial lining of the sinus and cyst walls may be largely replaced by granulation tissue with a foul smelling discharge persisting.

Incidence: It occurs in age groups from 15 to 40, although the majority of cases reported in the Army were in patients from 21 to 30 years old. (The majority of men in the Army fall into this age group). It is more commonly found in males in the ratio of 3 to 1. In the female it has a tendency to occur earlier in life than it does in the male. It is strikingly confined to the Caucasian race.

Symptoms and Diagnosis: It may be a painful or painless sinus or swelling usually located in the sacrococcygeal region. There is a complaint of foul smelling discharge, persistent or intermittent. The patient may have symptoms of a boil or abscess. The sinus is usually in the midline or laterally placed.

Differential Diagnosis: (1) Fistula in ano; by injection of dye into the sinus and using a rectal speculum seeing the presence of dye in the anal canal. (2) Sebaceous cyst; a history of swelling or a lump and when not inflammed it is movable. When the cystic area is opened a cheesy-like substance is found. (3) A

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simple cyst is movable, raised and not inflammed. (4) Osteomyelitis of the sacrum and coccyx; this condition is more painful and there is more systemic and pronounced local reaction. (5) The history of the patient and the X-ray are used in determining the presence of tuberculosis of the lower spine.

Prognosis: Medical (supportive) treatment is not curative. Radical surgery improves the number of good results, but recurrence rates as high as 20 per cent have been reported. The required period for healing as reported usually comprises 2 to 4 months.

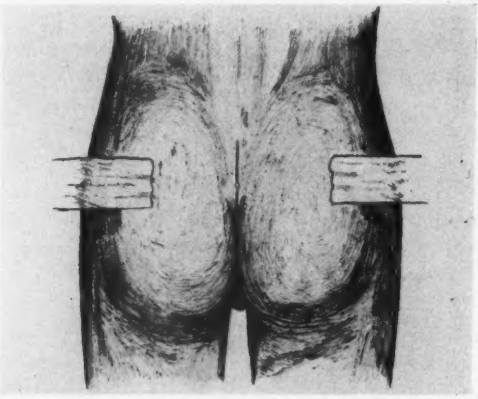

FIG. 1. Method of applying adhesive for better exposure of the sacrococcygeal region

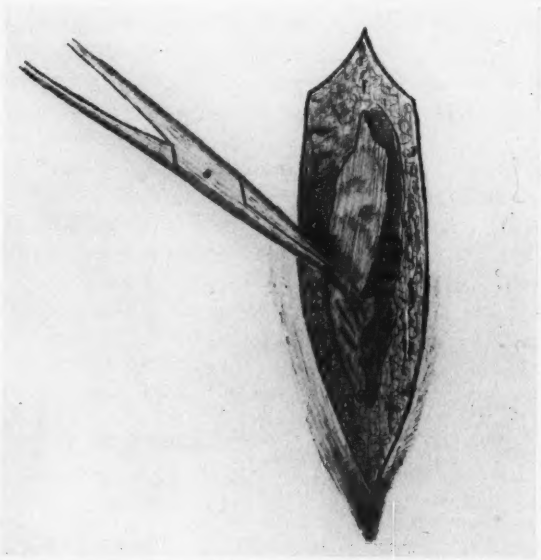

FIG. 2. Inclusion demonstrating marked inverted "V" at upper angle for excision of pilonidal cyst and sinus.

In reporting a series of operations in 162 cases of pilonidal cysts and sinuses, 77 cases were done at the Regional Hospital at Langley Field, Virginia, from July 18, 1942 to Jan. 31, 1944. These 77 cases were done by the primary closure and operative method as follows: After the removal of the sinus and cyst and skin en bloc, including the sacrococcygeal fascia, the gluteal muscles and fascia were undermined over the sacrum for 1½ to 2 inches on each side purposely everting the skin edges. The patients were given combined antibiotics such as penicillin and streptomycin with sulfonamides and sitz baths twice daily. The average hospital days were 26.24. Nine cases were done without muscle approximation but by removal of sinus, cyst and skin en bloc and not stressing the eversion of the skin edges. Primary closure was done. The average hospital days were 30.40. Five patients were incised and drained before operation, and the inflammation allowed to subside before radical operation with complete closure was done. The longest hospital stay was 52 days, the shortest 11 days.

When an abscess formation of the pilonidal cyst and sinus is encountered, it has been our experience that it is best to do a crucial incision and curette the pilonidal cyst and sinus tract along with the infected granulation areas. After curetting the infected cyst and sinus tracts, the skin edges are sutured down to the sacrococcygeal fascia and packed with iodoform gauze as advocated by Carrington.[1]

Operative Procedure: It is believed that the best results are obtained by making an elliptical incision with a sharp inverted "V" at the upper angle to prevent puckering when the wound is closed. Skin, sinuses, and cysts en bloc are excised down to and including a portion of the sacrococcygeal fascia. The gluteal muscles and fascia are undermined sufficiently to permit their approximation over the sacrum and coccyx without tension. Hemostasis is best controlled by suture ligatures, hot packs and pressure. The muscles and their fascia are approximated by interrupted no. 1 chromic catgut sutures bringing the gluteal muscles and fascia over the sacral and coccygeal regions by passing the sutures through the muscles and fascia including a small portion of the sacrococcygeal fascia. Muscles and fascia should not be sutured over the sacrum with any tension. The superficial fascia is closed with interrupted no. 00 plain catgut, being sure to have good hemostasis and approximation without tension and filling in all dead spaces with muscle and fascia. The skin is closed with no. 000 or no. 00 black silk mattress sutures purposely everting the skin edges. This method of closing is to prevent the downward migration of epidermal cells which Womack[4] believes is the cause of frequent recurring sinuses.

This method (1) shortens the healing time, (2) gives a pliable muscle pad over the sacrum, (3) prevents painful and large scars, and (4) closes all dead spaces. It has been noted in the majority of our cases, that a certain amount of numbness is experienced by the patient in the incisional area for several weeks following the operation. This is not permanent.

The muscle and fascia sutured over the sacrum has a tendency to absorb the serum-like collection of fluid in the base of the wound. We do not use methylene blue or any dye in injecting sinuses, because this has a tendency to distort and

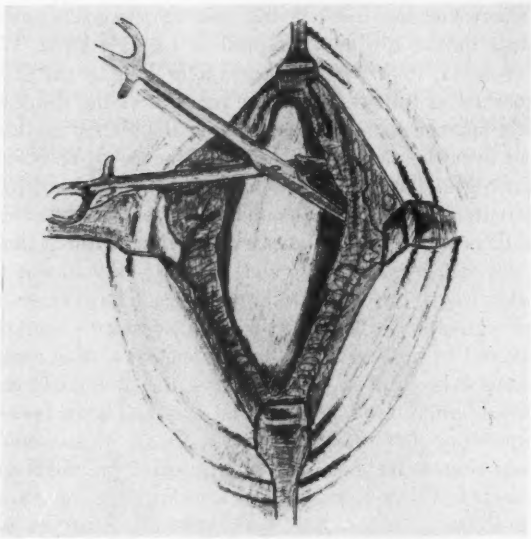

FIG. 3. Incision undermining gluteal muscles and their fascias from sacrum and coccyx

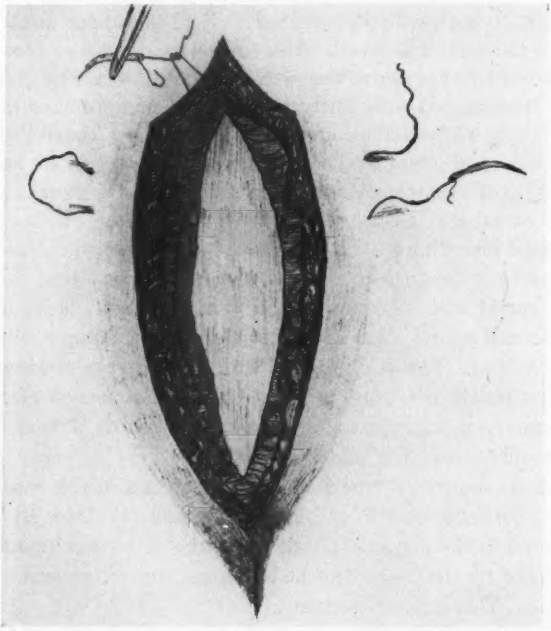

FIG. 4. Muscles and fascia approximated over sacrum and coccyx without tension. Mattress sutures to demonstrate eversion of skin edges.

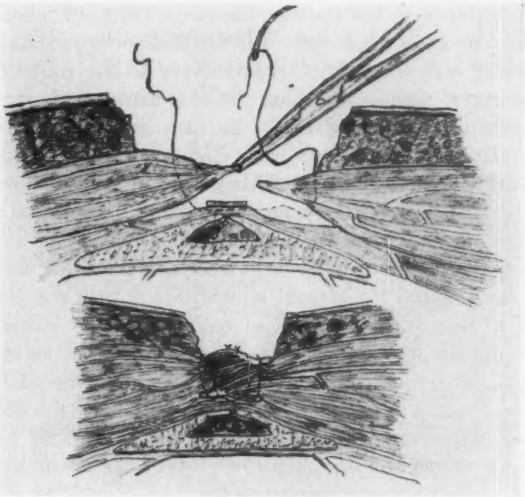

FIG. 5. Cross section of figure 4

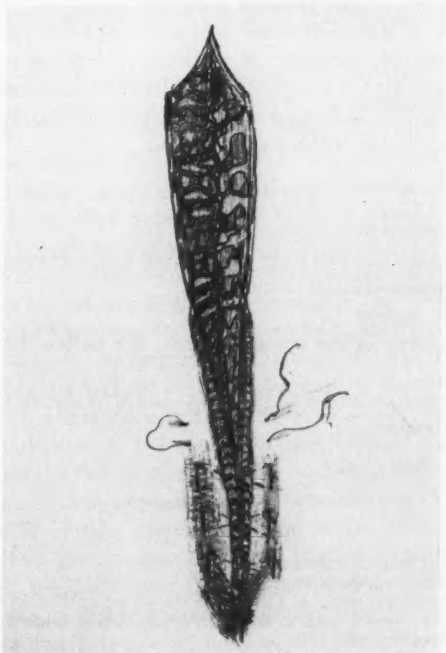

FIG. 6. Interrupted mattress sutures, purposely everting skin edges to prevent the downward migration of epidermal cells.

disguise the appearance of the tissues. The sinus tracts of pilonidal tissue are readily recognised by gross appearance if the pathologic appearance of the tissue is not distorted by a dye. All tracts are removed at the time of surgery. The sacrum and coccygeal regions including the anal area are thoroughly prepared and shaved preoperative, and the base of the incision is kept shaved from 2 to 3 weeks postoperative to prevent any hairs from growing back into the incision and thereby start another sinus tract. This procedure has cut down the hospital days and has given better results, eliminating painful scars and improving the cure rate. It is sound anatomically and physiologically. The average hospital days in Military Service were 26.24 days after which soldier patients were discharged from the hospital for general military duty with complete healing. In civilian life the average days from date of operation to date entirely healed were 12 and the average hospital stay was 5.6 days. From this series of 85 cases there have been only 2 recurrences. The so-called recurrence of pilonidal cyst and sinus is not due to the remains of pilonidal tissue. It is caused by not obliterating dead spaces; by poor hemostasis; by patients not being properly prepared before surgery; by not everting the skin edges to prevent the downward migration of epidermal cells, or by poor postoperative care.

Postoperative Care: 1. Patients are not restricted as to position in bed. 2. Patients are allowed out of bed the day after operation and encouraged to walk around their room. 3. Antibiotics and sulfonamides are used 4 to 5 days postoperative. 4. Liquid diet is given for 48 hours and bowel movements are not encouraged until the third or fourth day. 5. After 48 hours a regular diet is given if desired. 6. The incision is dressed on the third postoperative day and again on the sixth day when all skin sutures are removed.

CONCLUSIONS

This method shortens healing time.

It gives a pliable muscle pad over the sacrum and coccygeal regions.

It prevents painful and large scars.

It closes all dead spaces.

Because of numbness in the operative area, the patient has less pain during convalescence.

The gluteus maximus muscles and their fascia aid in absorbing serosanguineous fluid and also aid hemostasis.

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THE TREATMENT OF DEPRESSED FRACTURES OF THE SKULL WITH SPECIAL REFERENCE TO THE REPAIR OF THE CRANIAL DEFECT

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Whenever there is a depressed fracture of the skull in connection with a head injury there is practically always an injury of the dura and brain directly underneath the fracture, and sometimes a diffuse brain injury at a remote distance from where the blow was received. We are now concerned primarily with the injury of the skull and the repair of the skull defect; however, one cannot overlook primarily the treatment of the underlying soft tissue injury of the brain and dura. One has to take into consideration whether or not the depressed fracture is simple or compound, the difference being that the latter is a contaminated wound and should be treated surgically during the first 24 hours, while the simple depressed fracture can be operated upon several days later provided the patient's condition and symptoms of compression are not too great. It is very important that the cranial contour and integrity be maintained as far as possible. A brief discussion will be made of different methods of cranioplasty used to repair the cranial defect.

Depressed fractures of the skull are usually caused by some sharp or protruding object striking the head as may be caused by an axe, hammer or projecting bolt or similar object from a moving vehicle. Most frequently there is a laceration or break in the contour of the skin overlying the fracture, and it is not unusual that the emergency room surgeon may suture the laceration of the scalp allowing the depressed fracture to be overlooked without first making a thorough examination of the cranial contour with the sterile glove finger or an instrument through the open wound. More frequently these patients are not rendered unconscious because the local break and depression of the bone takes away the concussion effect of the blow, and there may not be widespread brain concussion from the injury. Too often the laceration is sutured and the patient is sent out of the hospital because he had not been unconscious. If there is any doubt that there may or may not be a fracture or a depressed fracture underneath the laceration, an x-ray examination of the skull should always be made which should show the fracture and the degree of depression. Should there be leakage of spinal fluid or lacerated brain tissue appearing in the wound, it is pathognomonic evidence that there is laceration of the dura and brain usually with depressed fracture of the skull. If the examining surgeon is sure that there is a depressed fracture underneath the laceration, there is no need to apply sutures to the scalp in the emergency room but instead the patient may be taken to the operating room for surgical repair. However, there should be wide shaving of the scalp

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and proper cleansing with salt solution or suitable antiseptics. If there is to be any delay in surgical repair in the operating room, the wound may be closed with sutures to close the wound and stop bleeding. A delay in the operation may be required because of the associated injuries and poor condition of the patient. The delay should not be longer than necessary and the operation preferably should be done during the first 24 hours. During the period of waiting adequate antibiotic therapy should be instituted.

The chief difference between a simple and a compound depressed fracture of the skull is the slight increase of danger of infection in the wound in the case of the compound fracture, while in the simple depressed fracture the delay may be for several days. Most of these patients who are adults and cooperative can be operated upon under local anesthesia while children and semiconscious, unruly patients may require a general anesthetic. The skin is prepared in the usual manner with suitable antiseptics following shaving. The ragged edges of the laceration of the scalp should be excised with a sharp knife which is then discarded so as to convert the wound into a clean incised wound. It may be necessary to extend the edges of the incision to get adequate exposure of the underlying fractured area. It may be necessary to put in a drill hole large enough to admit a small periosteal elevator to elevate the bone fragments. One may have to use a narrow pointed bone rongeur to bite away the edges of the fractured line to free the impinged depressed fragments. The depressed fragments from the inner table of the skull will cover a wider area than that of the outer table. It is sometimes necessary not only to elevate but remove completely some of the comminuted fragments of bone, but they are saved in saline solution for possible replacement and repair of the cranial defect. It is necessary to search for and remove loose fragments of bone from the inner table found lying between the dura and the skull in the neighborhood. A lacerated dura and brain should be thoroughly inspected and irrigated with Ringers solution to remove loose pulpified fragments of brain, pieces of bone and blood clots.

If the dura has not already been opened and it is tense and bluish, it should be opened for a fraction of an inch or sufficiently to inspect the subdural space for blood clots and their removal. The dura should always be sutured tightly with interrupted fine silk sutures. Sometimes in severe extensive depressed fractures of the skull there may be extensive lacerations of the dura, merely torn into shreds. If one is careful to spread out the torn membrane, one may find enough dura to effect a suitable closure. However, if a tight closure of the dura is not possible one may get some fascia from the temporal muscle, from the galea, or from the fascia lata.

An unclosed dura may increase the danger of herniation of the brain leading to a brain fungus and a possible brain abscess. This danger is greatly lessened with a tightly closed dura should an infection occur in the scalp wound. To repair the cranial defect made in the bone, the elevated fragments of bone may be pressed back to the normal level of the skull edges. The bone fragments previously removed are cleansed and sterilized in antiseptic solution, and reshaped with bone rongeurs to fit into the cranial defect to lie at the normal level of the skull.

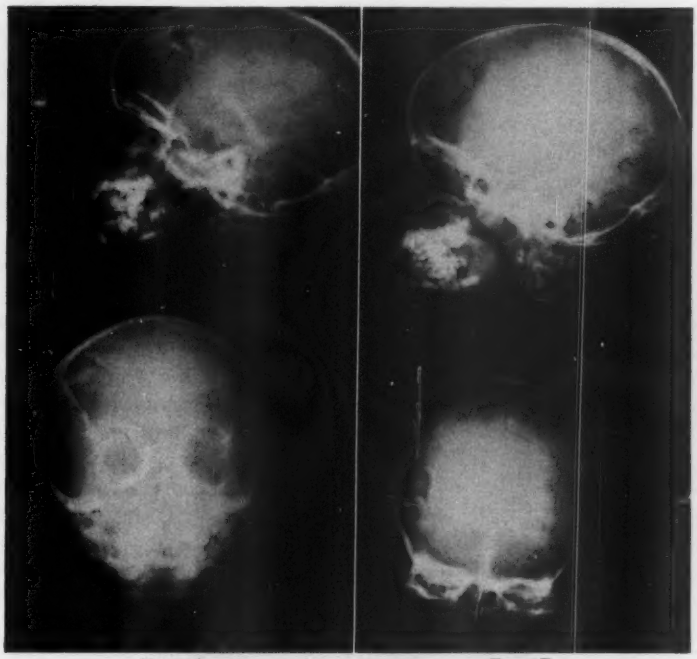

FIG. 1A FIG. 1B

FIG. 1A. Roentgenologic examination. Compound depressed fracture skull right frontal region, Aug. 11, 1956. B. Roentgenologic examination. Postoperative repair, Oct. 12, 1956.

A multiple number of these bone fragments may be replaced to fill in the defect. It is not necessary to wire them in place since the normal contour of the underlying dura with the intradural pressure will keep them lifted outward to maintain the usual contour of the skull. The scalp wound is then sutured over the fractured area using two layers of interrupted silk sutures, for the galea and the skin. It is important to have the wound as dry as possible and not use drainage. In compound wounds the patient should receive large doses of antibiotics for several days to prevent infection.

It is realized that this method of treatment of compound depressed fractures of the skull may be somewhat at variance to the teaching in some clinics. It has been the custom to remove loose fragments of bone in the case of a compound depressed fracture of the skull, which may leave a cranial defect in some instances covering a very large area. The author has practiced the replacement of bone fragments to act as grafts and to maintain the contour and the integrity of the skull even though these defects may be large in extent. This practice has been used in a large number of cases, over an extensive period of time even before the days of antibiotics. Coleman[2] was an advocate for the replacement of bony fragments in simple depressed fractures of the skull, but he hesitated to replace them in cases of compound depressed fracture. It has been proved many

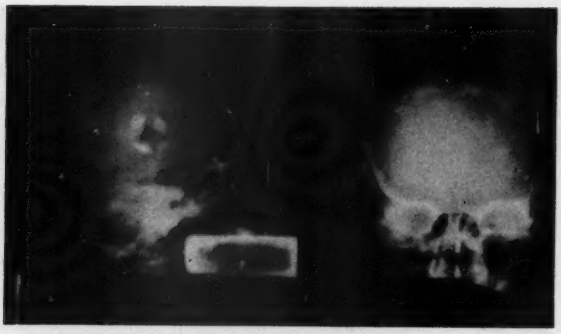

FIG. 2A

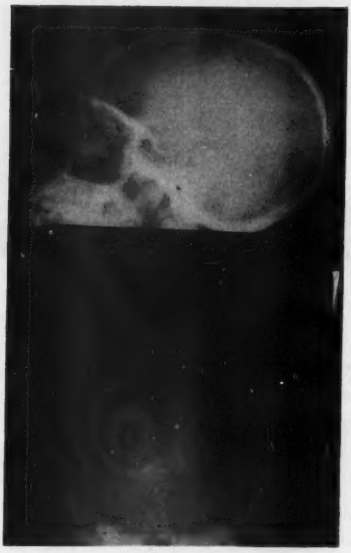

FIG. 2B

FIG. 2A. Roentgenologic examination. Compound depressed fracture of skull left temporal region before operation on April 28, 1953. B. Roentgenologic examination. After operation on May 7, 1953.

times that these fragments of bone can be replaced in compound depressed fracture of the skull when operated upon a few hours after the injury, and with antibiotics a delay of 16 or 24 hours may represent a safe period for the operation.

It is better to repair the cranial defect at the time of the initial operation by using living bone tissue for that purpose. The patient will feel better in knowing

that he has normal living bone over the fractured area rather than a metal plate or other foreign substance in his skull.

Other materials can be used for plastic closure of these cranial defects with good results which are relatively inert in contact with living tissues. Tantalum[4] plate is one of these satisfactory materials which can be shaped, molded and fashioned to the outer table of the skull but it requires considerable hammering, shaping and fitting which is time consuming in the operating room. Furthermore, the plate when placed over the defect, or at a level with the outer table of the skull, may permit a dead space between the dura and the plate as much as the thickness of the skull. However, this is not a serious objection, and it is probably more of a theoretical objection than one that may cause any harm. Another satisfactory material is Methyl Methacrylate.[3] It is a resin material and can be molded and fitted in the cranial defect in a relatively short period of time in the operating room. It has the advantage of replacing the usual normal thickness of the skull thereby preventing a dead space beneath the plate. It is impervious to x-rays thereby not interfering with future x-ray examinations such as encephalography, ventriculography, and arteriography. With Tantalum plate this objection may be a real one since it would interfere with some x-ray examinations.

A third very satisfactory material is polyethylene[1] plate, which is inert and innocuous to tissues. It can be molded in proper shape and cut to fit the defect

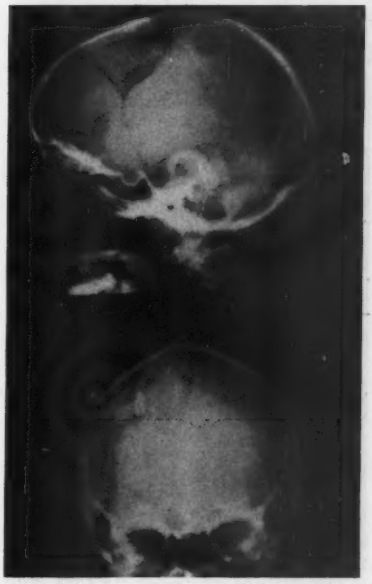

FIG. 3. Roentgenologic examination on Feb. 6, 1957 of right frontal region, compound comminuted depressed fracture of skull operated on with preservation and replacement of bone fragments on Aug. 15, 1939.

in a short period of time in the operating room at the time the cranial defect is to be repaired. It is also impervious to x-rays and does not interfere with future examinations of this type. Celluloid[2] plate has been used over a long period of time and was the material of choice before the above mentioned plastic materials were developed. The advantages of celluloid plate were the easy molding and fitting in the cranial defect without interference in later x-ray examinations. However, it is not as innocuous to tissues as the above mentioned materials and it does cause a certain amount of reaction and sometimes requires removal. With the above mentioned materials available it is doubtful that celluloid plates will be used anymore.

Autogenous bone graft material may be used over a smaller cranial defect but it is usually not satisfactory to cover a large cranial defect. The chief objection to a bone graft is that it may require an additional surgical incision if the bone is taken from the tibia or a rib. Furthermore, bone from these areas is of a different type, having a periosteum which may produce an overgrowth of bone, thereby interfering with the cranial contour or cause an ingrowth of bone intracranially. An autogenous bone graft from the outer table of the skull taken in the neighborhood of the cranial defect is bone laid down in membrane and does not produce an overgrowth of bone. However, this is a time consuming operation

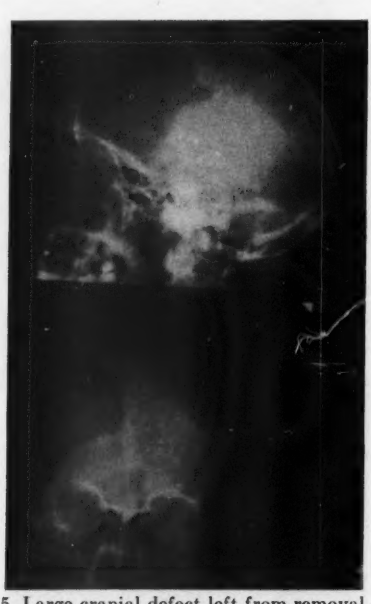

FIG. 4. Patient aged 45. Large cranial defect left from removal of bone fragments when operated on for compound comminuted depressed fracture skull, right frontal region in another city in November 1953. In September 1955 satisfactory cranioplasty done using Methyl Methacrylate. If bone fragments could have been replaced the latter may not have been necessary.

and it may be too strenuous and severe for the patient, so that the use of one of the above plastic materials may be preferred.

CONCLUSIONS

Compound depressed fractures of the skull should be operated upon as soon as possible to prevent infection.

Secondarily, simple and compound depressed fractures of the skull should be operated upon as soon as possible to remove depressed fragments of bone, blood clots and foreign material, and to repair the dura and the brain.

The bone fragments should be elevated and loose detached bone fragments saved and replaced in the defect to act as a bone graft and preserve the contour and integrity of the skull. This can be done with greater safety in compound wounds with the use of antibiotics.

Other materials may be used for cranioplasty, primarily in simple depressed fractures, and secondarily in compound depressed fractures, such as tantalum plate, methyl methacrylate resin material and polyethylene plate material.

Some type of bone graft may be done later to cover over the cranial defect, but it is better to fill in the defect with the patient's own bone fragments recovered at the original operation.

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PITFALLS IN THE MANAGEMENT OF CANCER OF THE HEAD AND NECK (ORAL CAVITY)**

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Progress has been made in the management of cancer of the oral cavity in the past 50 years as the result of improved technics in radiation and surgical treatment, better anesthesia, and better postoperative care. These have brought about a decline in the postoperative and posttherapeutic mortality and to a lesser degree in morbidity. Despite all of this, we have not yet achieved the desired end result, namely, the saving of lives in the majority of those afflicted by this disease.

If we analyze 100 cases, we find that approximately one-third of these are immediately cast aside as the incurable group despite any heroic measures that may be performed. This leaves us with two-thirds of the patients to treat, and of this number, only one-half or less survive 5 years free of disease.

We have had the same experience, both good and bad, that you have encountered. Being dissatisfied, we have paused to reflect and to try and rationalize our approach to this problem. We will talk of some of these thoughts which seem sound to us, be they right or be they wrong, only time will tell.

The life of the individual therapist, be he radiologist or surgeon, is too short; he has too small an experience to evaluate all forms of therapy. Indeed, even in large centers, this causes difficulty. The final answer now is not available on long term survivals for different radiologic modalities and surgical procedures.

We have selected epidermoid lesions of the tongue, lip, floor of mouth, gingival margin, and buccal mucosa, as they present similar, and comparable diagnostic, and therapeutic problems. We will discuss pitfalls encountered in the management of these lesions.

The selection of therapy certainly poses a pitfall. Ogilvie[13] stated: "To fall into a pitfall is humility to the surgeon, but it may be disastrous to the patient. We should all know our normal anatomy. Surgery is the art of doing something and to be effective, that something must be done at the right time."

One of the first major problems is the failure to fully appreciate the biologic potential or natural behavior of the cancerous process and the mode of cancer dissemination. It may be superfluous to review briefly the spread of cancer, but it does reveal the difficulty encountered in assessing the decree of local extension and possible lymph node involvement. This is in part explained by the manner in which cancer cells invade tissues, lymphatics, and vascular channels. Tumor

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cells extend by infiltration of tissue spaces, by dissecting the tissues apart along planes of anatomic cleavage, and by occupying all available space.[22] For instance, one may find clumps of malignant cells in immediate contact with muscle fibers. The extension into tissue spaces determines more or less the size and the shape of the tumor, the degree of induration, and its mobility.

From the tissue spaces, the tumor may then invade the lymphatics and vascular channels. Tumor emboli gain entrance into the lymphatic channels and spread to lymph nodes. There are two very important factors concerned with the dissemination of cancer by the lymphatic system. One is the fate of the tumor emboli, for many metastatic emboli either perish or remain sterile in their new site of arrest. Other emboli, of course, go on to increase in activity, however, we do not know why tumor cells prove fertile in certain areas and remain barren in others. The second factor is the fact that tumor emboli travel in lymphatic channels to the lymph nodes, but so far no one has seen cancerous lymphatic vessels connecting the primary with the secondary tumor. Lymphatic dissemination from the initial lymph node deposit is often extensive. The tumor is transferred from gland to gland until a group or large part of the entire lymphatic system is involved. Furthermore, the occlusion of lymphatic vessels by tumor cells may result in the reversal of the normal direction of lymph flow and cause retrograde extension.

As for the spread of cancer of the oral cavity, one can usually predict the pathway of lymphatic spread. Lesions localized to one side of the midline usually spread to those on the same side, but they may spread to the opposite side.

The difficulty with dissemination of the process by the lymphatic system is to clinically determine when a lymph node, whether it be palpable or nonpalpable contains cancer. Too many times we assume that a nonpalpable node does not contain cancer and we therefore delay definitive treatment and await lymph node palpability.

Lesions of the oral cavity, particularly those of epidermal origin, are confined above the clavicle. It is estimated that 80 per cent of the lesions remain confined to this area, therefore, these lesions are accessible for diagnosis and therapy.

Carcinoma of the tongue produces lymph node metastasis early in a high percentage of cases, at least 80 per cent in fatal cases (Willis[22]). In cancer of the tongue, cervical lymph gland metastasis may predominate on the side opposite that of the primary lesion and sometimes the contralateral metastases are the first to appear. This is in conflict with Ackerman[1] who stated that the contralateral metastasis does not occur until after homolateral metastasis has developed. Not infrequently, these metastases are the earliest clinical signs of the disease.

Carcinoma of the lip produces glandular metastasis more slowly and in fewer cases.

According to Royster,[18] in a study of cervical lymph node metastasis in squamous cell carcinoma of the oral cavity, it was found that in the suprahyoid dissection there was an average of 8 lymph nodes per specimen and for the infrahyoid area there were 36 lymph nodes per specimen. According to Rouviere[16] the total number of nodes on one side of the neck in the area ordinarily removed in a radical neck dissection has been found to vary from 23 to 48.

If cases are further analyzed as to the presence of metastatic involvement of lesions from the oral cavity when first seen, it is noted that reports in literature by various authors, such as Copeland,[6] who stated that patients, when they first present themselves for treatment, positive cervical lymph nodes will be found as follows: lip 4–30 per cent; tongue 35–60 per cent; gingival margin 26–65 per cent; buccal mucosa 30–50 per cent; and floor of mouth 25 per cent.

Another very important point of consideration is that of the "forgotten zone" as described by Pressman.[15] He calls attention to the chain of lymph nodes underlying the ribbon muscles above and below the thyroid cartilage and the hyoid bone.

Another area of concern to us is that of the passage of lymphatics through the mandible. Polya and Navratil[14] demonstrated that in 50 per cent of patients, the lymphatic vessels of the tongue and floor of the mouth pass through the periosteum of the mandible on their way to the upper cervical nodes. Byars[5] has stressed the involvement of the mandible via the mental foramen in adjacent cancers of the lower lip, gingival margin, and floor of the mouth. In elderly edentulous patients, the bone absorption causes the position of the mental foramen to become quite superficial and vulnerable for easy invasion by adjacent cancer. He also points out that in cancer of the lip, it at times metastasizes to the buccal node, which when involved, quickly attaches itself and invades the mandible.

Having determined the full extent of the primary and the probability of lymph node involvement, a decision must be reached regarding the type of therapy to be employed, whether it be radiation or surgery. Questions come to mind as to the operability of the lesion and the surgical risk of the patient. Martin[12] says that there is no medical contraindication to cancer therapy.

The selection of therapy indeed poses a pitfall. The prime objective in curative cancer therapy is control of all of the tissue involved with cancers, that locally, that in the regional nodes, and that in the intervening lymphatics and tissue.

The philosophy of the therapist and the crystallization of his concept about cancer therapy is important in dealing with the patient's condition. He must be willing to realize that some deformity will exist, and will he be willing to accept this as well as some unnecessary procedures? He must also realize that radiation too, has mortality and morbidity, even when judiciously administered. He must be aware of his therapeutic ability and his genuine interest in the problem. He must be willing to devote all the time that is necessary, for frequently there is a large investment of time in the treatment of these patients. He must be willing to accept failures, for there will be many cases with morbidity, mortality, and complications. Without a rather firm personal concept regarding this, the therapist probably cannot render as good judgment in the handling of these patients.

All of our cancer cases are reviewed by the Tumor Board, which is composed of representatives of all pertinent departments. We believe that the evaluation is better in these cases when it results from decisions and discussions from the radiologist, pathologist, surgeon, internist, dentist, and those interested in re-

habilitation. The dentist plays an important role for it is he, when properly orientated, who detects most of the intraoral lesions early. When he pursues an unknown lesion with an immediate biopsy, a great service is done to the patient. It is obvious that the best results in these cancer cases are accomplished, for the most part, in the smaller, earlier lesions. He is also aware through his orientation, of the significant incidence of second carcinomatous lesions within the oral cavity. He aids materially in the preoperative planning regarding any prosthesis, which will be used in the postoperative phase for cosmetic purposes or therapeutic carriers of radiation. Likewise, he controls poor intraoral hygiene and sepsis before intraoral cancer therapy is begun.

Control of local lesions often may be accomplished by either radiation or surgery if vigorously and properly done. More important than the choice of radiation or surgery is the ability and attitude of the respective therapist. If the choice of the treatment would yield a near equal result and the radiologist has much experience and interest, it is obvious that he can do a better job than the surgeon who is not interested in these conditions. Likewise, not all radiologists are interested or familiar with these various technics.

Reports from the behavior of radiation therapy for lesions of the lip, tongue, the gingival margin, and the floor of the mouth offer hopeful results, but its effectiveness is limited to localized lesions. Radiation has little to offer in the routine control of lymph node metastases.

The eradication of the local lesion by radiation followed by node dissection ignores the intervening lymphatics and tissue which might contain cancer. For this reason we favor incontinuity dissection of the local lesion along with the lymph node bearing area. Long term results with the commando, pull through or segmental resection are insufficient in number to unequivocally call this the treatment of choice.

With our present limited knowledge we must treat these patients intensively if we wish to salvage the most. Our first therapeutic effort offers the best chance. In doing so, some unnecessary therapeutic procedures are going to be performed on some patients; however, without this approach some patients are going to be lost who could have been saved.

Lymph node metastasis is common with oral cancer. According to Ackerman[2] 40 per cent of the patients with cancer of the tongue, when first seen, will have positive nodes. Another 40 per cent without positive nodes on the first visit will later develop cervical metastasis even after control of the primary lesion. Because of this high incidence of involvement, we agree with those advocating prophylactic neck dissection for the above mentioned oral cancer, save that of the lower lip, and even here in some few circumstances.

Some authors, like Martin,[10] do not favor prophylactic neck dissection, but others like Royster[18], believe there is a need, as there is no method of determining preoperatively whether the cancer is present in lymph nodes that are not palpable, especially in the deep cervical region where the cancer of the tongue usually metastasizes first. In order to remove nonpalpable cancerous lymph nodes a prophylactic neck dissection must be done.

The watchful waiting approach to the treatment of nonpalpable lymph nodes seems to us to be a major pitfall as well as a so-called "node picking" and other localized resection technics of suspected nodes.

Indeed we subscribe to the practice of staged, prophylactic, contralateral neck dissection under most circumstances, if the unilateral nodes are positive and if the lesions involved are near the midline. Strictly unilateral carcinoma of the anterior two-thirds of the tongue involves contralateral nodes in 6 per cent of the patients, whereas those close to or beyond the midline have a 32 per cent involvement (Lyall[9]).

It is not within the scope of this paper to review all the surgical procedures that have been employed in the past in the surgical removal of lesions of the oral cavity, but it is well to mention a few of these: The radical neck dissection first described by Crile;[7] the pull through as described by Ward and Hendrick,[21] and later by Slaughter;[19] the division of the jaw to provide access to the intraoral lesion by Roux[17] and Billroth,[4] and later by Kremen;[8] the segmental resection of the mandible and finally the combined operative procedure known as the neck dissection, hemimandibulectomy excision of the primary tumor as ascribed to Martin.[11]

Incontinuity dissection frequently allows for more radical excision of the local tumor and eradication of the intervening tissue from the local sites to the area of possible lymphatic spread. This tissue may contain tumor by spread by the lymphatics or by direct continuity. In addition, mandibular section often affords more adequate exposure for relatively nonmanipulative resection of intraoral cancer. The added exposure will also permit the therapist to better evaluate the local nature and the extent of the lesion, thereby aiding in more complete control.

Another pitfall is the question of the spread of tumor as a result of surgical manipulation. This has been emphasized by Ackerman[1] and Smith.[20] The examination of material from surgical wounds has revealed positive identification of tumor cells in 28 per cent of cases, as reported by Smith.[20]

Such radical surgery is meticulous and time-consuming, and is another pitfall because of surgeons' fatigue. Cooperative effort enters into the treatment by using two teams or parts thereof, in order to give transient respite during these lengthy procedures. We believe that the patients have benefited by the sustained care, judgment and technic of the operating personnel. Likewise, the surgeon has profited by less mental and physical fatigue.

In our opinion the best results may be obtained from the continued procedure of primary excision of the tumor together with the intervening tissue and radical neck dissection, including the "forgotten zone". This may also include the mandible, the tongue, floor of mouth, buccal mucosa, and gingival margin. We are becoming more and more radical with earlier lesions, believing that the morbidity and deformity are not too heavy a price to pay.

If the patient is to have a deformity, such as the loss of half a tongue, or half of the mandible, we can help him by taking time with explanation and orientation and can aid him considerably by having a similarly treated and recovered patient

visit him. Such cured patients who have made reasonable adjustments in their return to society will do much to help our patient with his decision, recovery, and rehabilitation.

All of the patients who have advanced cancer cannot be cured and no doubt certain therapeutic procedures probably hasten death, as well as increase the morbidity and complications. Unfortunately, we cannot predict the salvageable advanced cases with any degree of accuracy, and our judgment has often been wrong.

However, we have a decided responsibility to the patient we have treated unsuccessfully. The patient who is aware that he is going to die from his cancer, not only has a physical morbidity in the complication of his disease and therapeutic failures, but he has psychic morbidity also. His main fear is often not that of dying, but that of being forgotten by his therapist, now that the patient has an advanced, incurable disease. He wants to know that his physical suffering will be cared for to the end. We can anticipate this and assure the patients of our continued interest and actually follow this through with regular follow-up visits for examination and treatment. Under these circumstances, the patient's mind will be kept more free for much more important mental adjustment during his remaining life.

"In cancer therapeusis the primitive doctrine of kill or be killed is especially true. Action must be swift, decisive, and heroic. To temporize is to surrender without battle. To economize in the scope of our surgical or radiologic attack is to fall short of success. To consider post-therapeutic deformity is to lose the battle. Here most certainly the more one saves the part the more surely one has lost the whole." (Anderson[3])

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EXCISION OF CAROTID BODY TUMORS

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Carotid body tumors are uncommonly encountered and the present literature contains less than 400 reported cases. Although considered to be tumors of the chemoceptor system, their systemic effect is rarely encountered. These tumors grow slowly and most symptoms are due to local effects of pressure upon vital adjacent structures. Harrington and associates[2] indicated that 30 per cent of these tumors were malignant but present day criteria have established few instances of malignancy and metastases from these tumors.

These tumors originate at the carotid bifurcation. They are intimately adherent to the carotid arteries and are quite vascular. The reports of complications and deaths from removal of carotid body tumors stem predominately from ligation or injuries sustained by the common and internal carotid vessels. The preservation of these vessels has been stressed by Farrer and associates[1]. This is re-emphasized in the following case presentation.

CASE REPORT

A 47 year old white housewife was admitted to the hospital from a cancer clinic with a 12 year history of a slowly growing lump in the left neck. The patient remarked that it increased slightly in size when upper respiratory infections were present and decreased in size following antibiotic therapy. Since February 1957 the mass had increased more rapidly in size. Local pain radiated into the left lateral facial area and into the left retro-orbital area. This pain had recently awakened the patient from sleep and required narcotics for relief.

Examination revealed a slightly obese white female with a blood pressure of 120/80; a pulse of 74; respiratory rate of 18 and a temperature of 98.8 F. Dental caries were present in the lower teeth. Nasopharyngoscopic examination was within normal limits. A slightly movable, slightly tender, firm, ovoid mass was partially covered by the anterior margin of the sternocleidomastoid muscle just below the angle of the left mandible. Bruit and thrill were absent. A pulsation was transmitted from the carotid vessels. Other findings were chronic cervicitis and a 1° cystocoele.

Laboratory: urinalysis and blood counts were normal. A chest film and electrocardiogram were interpreted to be within normal limits.

On July 3, 1957 under general endotracheal anesthesia a submandibular incision exposed a 5 by 3.5 by 3 cm. reddish-blue, vascular, firm tumor in the carotid bifurcation and encircling the common, external and internal carotid vessels. The main tumor mass was placed medially (fig. 1). The vagus nerve was flattened posteriorly and was reflected without difficulty. The hypoglossal nerve was identified and preserved just superior to the tumor mass. The common and internal carotid arteries were isolated and tapes passed about them. A mobilization stitch was placed in the tumor mass for retraction. The external carotid artery was divided at the mandibular rim and reflected posterolaterally. The common carotid adventitia was incised and reflected. At the thinnest point the tumor was

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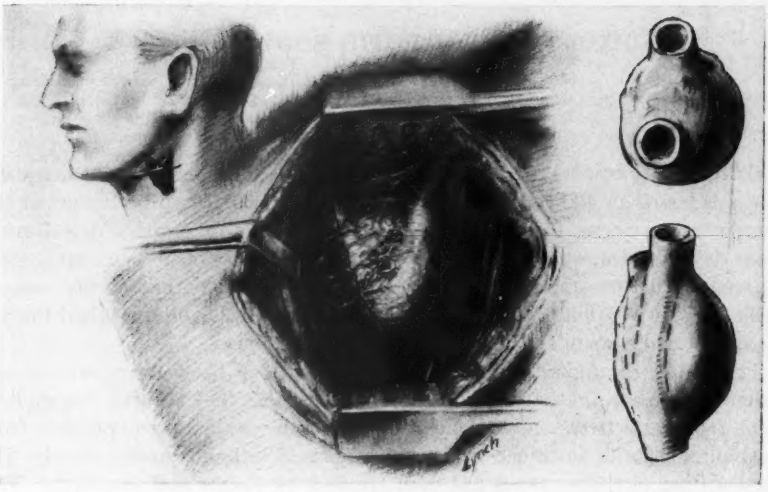

FIG. 1. Artist's conception of the tumor. The vagus nerve is posterior; the hypoglossal nerve is superior. The solid line indicates the incision into the tumor during removal. Inserts show the skin incision and the tumor's relationship to the carotid vessels.

divided between clamps after initially ligating and dividing the vascular bundles feeding the tumor. A dissection plane was readily accessible between tumor and arterial media. The tumor was removed without incident. Spasm of the internal carotid artery was controlled by adventitial infiltration with 1 per cent procaine and by immediate stellate block with 1 per cent xylocaine infiltration. Routine closure was performed without drainage. The patient received priscoline postoperatively and complained only of an easily controlled, retro-orbital headache. She was discharged on her third postoperative day without sequelae. The pathology report confirmed the diagnosis of carotid body tumor.

DISCUSSION

Division and reflexion of the external carotid artery greatly facilitated the operative procedure. However, in retrospect this tumor could have been excised without division of the external carotid artery. Had the distal external carotid been injected with heparin, reanastomosis after division could easily have been accomplished. When divided, the external carotid should eventually be ligated at the carotid bifurcation to prevent retrograde internal carotid thrombus formation. A prepared homograft or plastograft should be available either for direct insertion or as a by-pass shunt when segmental excision is anticipated.

Meticulous and patient dissection with early ligation of the vascular bundles feeding the tumor and discrete use of suction facilitates carotid body tumor excision. Prophylactic stellate block and postoperative papaverine or priscoline diminish arterial spasm.

SUMMARY

A patient is presented with a carotid body tumor complicated by pain. The surgical intervention is described.

Preservation of the arterial continuity can be accomplished by meticulous surgery. Reanastomosis of severed vessels directly or by graft insert should be accomplished. Operative stellate block and postoperative spasmolytic drugs should be administered to prevent internal carotid arterial spasm and subsequent thrombosis.

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NEWER DIAGNOSTIC METHODS IN THYROID DISEASE

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As late as 1956 Beierwaltes[2] stated that in its present form the determination of protein-bound iodine (PBI) was not easily adaptable to the clinical medical laboratory of the average community. His excellent paper, however, makes the obvious point that the PBI determination is a valuable tool for the clinician's use in determining the status of the thyroid patient. This chemical procedure when used in conjunction with the I^{131} uptake study will aid the physician in arriving at a proper diagnosis in those cases which defy clinical judgement.

Recent publications[1,3,7] have given laboratory directors improved and reliable methods for the determination of protein-bound iodine or serum precipatable iodine (SPI). The PBI blood level is today in many areas an available and, more important, accurate laboratory procedure. The widespread distribution of isotope laboratories, however, has made this procedure more accessible and consequently better known to the practioner. Each method has its limitations and each has advantages which we will point out.

It should be understood that each procedure tests a separate function of the thyroid. Radio iodine determines the ability of the gland to store iodine. Thus, in an overactive, hyperthyroid state high counts are recorded and the reverse is true in hypothyroidism. The PBI test determines the amount of circulating thyroxin as measured by the blood iodine. This chemical procedure tells us of the ability of the gland to form the hormone, thyroxin. High levels above 8 micrograms per 100 ml. of blood indicate hyperthyroidism and levels below 4 micrograms are indicative of hypothyroidism. In hyperthyroid patients after diagnostic I^{131} is given 2000–3000 counts per min. are recorded at 2 hours. Urinary excretion curves are 0–44 per cent. In the euthyroid state 700–1400 counts per minute are recorded at 2 hours with urinary excretion results being 44–70 per cent. In the hypothyroid patient there is slow uptake, 150–200 counts per minute slowly rising to 1200 counts per minute in 24 hours. Urinary excretion rates are 70–100 per cent.

It has been pointed out that these tests do not measure exactly the clinical disease of hyper- or hypothyroidism. That is, the effect of cellular stimulation by thyroxin cannot be judged by I^{131} or PBI studies alone. Yet, extensive use of these diagnostic aids has shown that as an index of formation and release of thyroid hormone they are generally accurate.[2]

Since these procedures are coming into more frequent use it would be well here to point out sources of error. Iodine administered before performing either uptake studies or serum PBI determination is the most frequent source of false

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results. Inorganic iodine sources are obvious: Lugol's solution, thyroid extract, cough medicines, etc. These can be easily controlled by keeping the patient from these drugs for a few weeks prior to testing. Unfortunately, the greatest difficulty in each test comes from the organic iodine in x-ray contrast media. This includes media used for cholecystography, intravenous pyelography, myelography and also for gastrointestinal series. The blood inorganic iodine rises rapidly after the

TABLE 1

Euthyroid patients

Patient Number	Clinical Diagnosis	I^{131}	Urinary Excretion	PBI
1	Hyperthyroid	Normal	60%	4.8 gamma
2	Hypothyroid	Normal	70	4.4
3	Hyperthyroid	Normal	59	4.5
4	Adenoma-Euthyroid	Normal	60	5.0
5	Hyperthyroid	Normal	50	2.9
6	Hypothyroid	Low	71	4.1
7	Hypothyroid-Post Op.	Low	86	5.1
8	Hyperthyroid	High	36	2.0
9	Adenoma	Normal	52	6.5
10	Hyperthyroid	Normal	55	12.7-artefact
11	Hyperthyroid	Normal	59	4.2
12	Hypothyroid	Normal	39	4.5
13	Adenoma	Low Normal	69	4.2
14	Adenoma-Euthyroid	High	52	7.1
15	Hypothyroid	Normal	70	6.0
16	Hyperthyroid	Normal	60	5.5
17	Hyperthyroid	Normal	46	5.2
18	Hyperthyroid	Normal	65	5.3
19	Hyperthyroid	Normal	40	6.2
20	Hyperthyroid	Normal	65	3.9
21	Hyperthyroid	Normal	60	5.4
22	Hyperthyroid	Normal	49	5.2
23	Hyperthyroid	Low Normal	66	4.2
24	Hyperthyroid-Adenoma	Normal	59	5.2
25	Hyperthyroid	Normal	64	5.0
26	Hyperthyroid-Adenoma	Artefact	65	16.8-artefact
27	Hyperthyroid	Normal	54	4.3
28	Hyperthyroid	Normal	64	6.1
29	Hyperthyroid	Normal	50	6.2
30	Hyperthyroid	Normal	52	5.8
31	Hyperthyroid	Normal	65	4.4
39	Hyperthyroid	Normal	62	3.7
40	Hyperthyroid	Normal	50	3.9
41	Hyperthyroid	Normal	61	11.1-artefact
42	Hyperthyroid	Low	80	11.7-artefact
43	Hypothyroid	Normal	63	3.4
44	Hyperthyroid	Normal	48	3.7
45	Hyperthyroid	High	40	2.0

Data from 38 euthyroid patients. Urinary excretion is expressed as percentage and PBI results are in micrograms per 100 cubic centimeters of blood.

patient receives these substances and this rise decreases the uptake of I^{131} by the thyroid. False values persist for at least 3 months. Obviously, the patient should be seen first by the clinical laboratory or isotopic medicine department before being sent on to the radiologist.

Many authors in the past have discouraged the routine use of the PBI test because of supposed technical difficulties rendering it almost impossible to perform. We have in our laboratories since 1951 done approximately 3000 PBI determinations first by the Barker method[4] and later by the Leffler method.[5] We have been tested for proficiency by a medical school laboratory[6] and found to differ by less than 0.28 micrograms. Our standard deviation is 0.44. We have not found it necessary to isolate the technician or to take extraordinary precautions with chemical storage or glassware. It must be understood, however, that the determination is to be done exactly and with above average technic since the amount of iodine in the final filtrate is 4 micrograms to 8 micrograms or gamma, a very minute amount which may be easily lost by a technical blunder. Our method of choice which may be done in any hospital with a competent technical staff and a well equipped laboratory has been published elsewhere.[5]

There are certain patients for whom the PBI determination may have definite advantages. The patient in congestive heart failure with rapid pulse which fails to respond may be suffering from unsuspected thyrotoxicosis. Moving a person in heart failure to the isotope laboratory may be impossible but a blood sample can easily be obtained by venepuncture and the PBI level be quickly done. Other patients may not be near an isotope center but the blood specimen can be forwarded by mail to the clinical laboratory. It may also be inadvisable to test the thyroid function of small children or pregnant women with isotopes but the simple venepuncture for PBI blood levels is not contraindicated.

TABLE 2

Hyperthyroid patients

Patient Number	Clinical Diagnosis	I^{131}	Urinary Excretion	PBI
32	Hyperthyroid	High	25%	13.0
33	Hyperthyroid	High	29	10.1
34	Hyperthyroid	High	31	8.9
38	Hyperthyroid	High	27.5	7.1

Data from 4 hyperthyroid patients. Results are expressed as in table 1.

TABLE 3

Hypothyroid patients

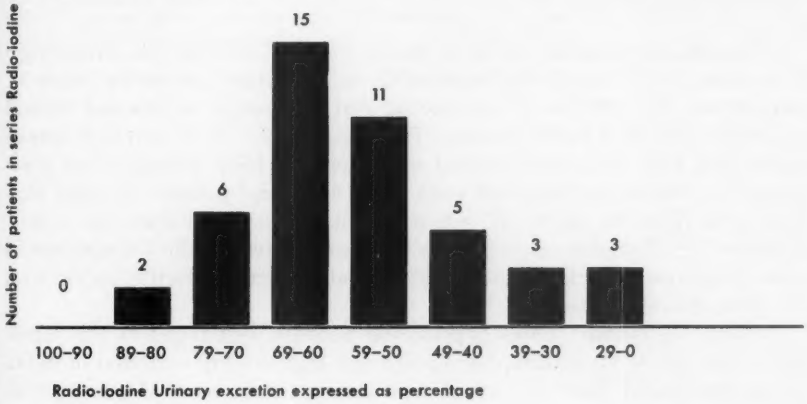
Patient Number	Clinical Diagnosis	I^{131}	Urinary Excretion	PBI
35	Hypothyroid	Low	75	2.7
36	Hypothyroid	Low	70	3.8
37	Hypothyroid	Low	78	2.0

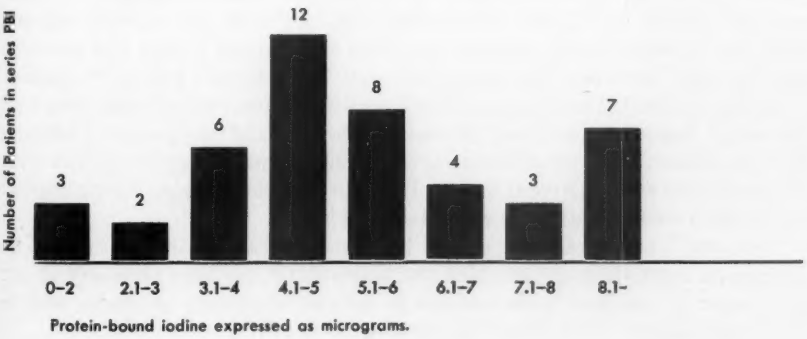
Data from 3 hypothyroid patients. Results are as expressed in tables 1 and 2

Advantages of the protein-bound iodine determination are that neither a fasting blood sample is needed nor must the patient rest prior to the test. The basal metabolism rate (BMR) is not considered an accurate procedure at best and is very inaccurate in the hyperactive apprehensive individual. The PBI is not influenced by those conditions other than thyroid disease which increase the BMR such as leukemia, hypertension, malignancies, Parkinsonianism and mental disturbances. The PBI is not lowered falsely by nonthyroid conditions which lower the BMR such as Addison's disease, Simmonds' disease and anorexia nervosa. The I^{131} study has the advantage of being a rapid method of estimating thyroid function as reflected in the gland's ability to take the iodine isotope into

TABLE 4

Results plotted against numbers of patients in each study. Excretion curves are expressed as percentages and PBI results are micrograms. The hypothyroid range is to the left in each bar-graph, the euthyroid range in the center and hyperthyroid range to the right. Note the close correlation between each group





its substance from the blood stream. There are many departments of isotopic medicine scattered over the nation and still only relatively few supervised laboratories performing the chemical test. This gap however is being rapidly narrowed.

Since the establishment of our Isotope program we have performed PBI determinations on all patients seen in the isotope laboratory at Wichita-St. Joseph Hospital. The data from 45 patients are presented in tables 1, 2, 3, 4. It is at once evident that in general the two methods parallel one another in results. Table 1 presents 38 euthyroid patients. Table 2 represents 4 hyperthyroid patients and table 3 presents 3 hypothyroid patients. In table 4 the 45 patients' results are shown in graphic form. The upper bar-graphy represents the urinary excretion rates as percentages and the lower, the PBI values are micrograms per 100 cubic centimeters of blood. The high values at 8.1 micrograms are due to several patients with iodine artefacts which have not been excluded from the compilation of results. Close correlation is shown between the two methods although separate functions of the thyroid are tested by each technic.

RESULTS

Hyperthyroid Disease: Among 45 patients tested 3 showed PBI levels above 8.0 gamma, the I^{131} uptake was high and the urinary excretion was low (table 1). One patient (No. 38) had a high normal PBI but isotope studies and clinical evaluation revealed hyperthyroidism. Two patients (Nos. 8, 15) had high uptake counts but PBI levels were normal or subnormal. Final evaluation of these patients indicated the euthyroid state. Four additional patients revealed high PBI levels (Nos. 10, 26, 41, 42) which were due to artefacts from oral iodine. In these the I^{131} studies confirmed the euthyroid states. Of the 4 hyperthyroid cases diagnosed 2 of these patients were treated with radioactive iodine with excellent clinical results.

Hypothyroid Disease: Three hypothyroid patients were found in this series. PBI levels below 4.0 gamma, low uptake and high urinary excretion of radio-iodine were found (table 3). One of these patients was a surgical hypothyroid. Two very low levels (Nos. 5, 8) were due to artefact from mercurial diuretics in cardiac patients whose thyroids were normal. Five low uptake studies were found (Nos. 7, 8, 13, 23, 42) with normal PBI and normal clinical findings. No hypothyroid patients were found with normal PBI levels. We have found that the PBI test is accurate in revealing unsuspected hypothyroid disease and errors in the low range occur less often than in the high. The reverse is true in I^{131} studies.

Euthyroid Cases: The remaining 38 patients were euthyroid although they had presented themselves to their clinicians with signs and symptoms of obscure thyroid disease. Basal metabolic rates and blood cholesterol levels together with physical signs were equivocal but the PBI and radio-iodine tests complemented one another substantiating the normal thyroid status.

From the limited series of patients it is evident that not in all instances do laboratory methods singly establish the diagnosis. It has been necessary in our experience to combine both technics in difficult diagnostic problems and to ultimately apply mature clinical judgement.

SUMMARY

In the relatively few thyroid patients who cannot be diagnosed by clinical evaluation the PBI and I^{131} determination will be definitive. The two procedures will produce best results if used together. Data from 45 patients' PBI and radioiodine studies are presented.

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GLASS BUTTON THORACIC DRAINAGE FOR CARCINOMATOUS PLEURAL EFFUSION*

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The use of the Crosby-Cooney button for carcinomatous pleural effusion was first reported by Doctor C. A. Alexander of Kalamazoo, Michigan in the American Journal of Surgery in August of 1953.[1] At that time he reported a case wherein the Crosby-Cooney button had resulted in satisfactory "spontaneous decompression" of the carcinomatous pleural effusion. The patient remained comfortable for the 4 months previous to her death without it being necessary to perform thoracentesis. At the time of autopsy there was a small amount of fluid (150 cc.) present in the pleural cavity. The button was patent corroborating the clinical evidence that it was functioning satisfactorily. Doctor Alexander reported in July of 1955 he had used the Crosby-Cooney button in 2 subsequent cases with good results. He reported that a colleague had reported a satisfactory result in one case.

To the 4 cases above reported by Doctor Alexander, the present report includes an additional 4 cases wherein the Crosby-Cooney button had been used for decompression of carcinomatous pleural effusion.

The surgical technic is that described by Doctor Alexander: "A U-shaped flap including skin and superficial fascia and deep fascia was reflected over the seventh, eighth and ninth ribs in the mid-axillary line. The periosteum of the eighth rib is incised and reflected for a distance of about 2″ and a section approximately ⅝″ long is resected and entrance into the pleural cavity is made through the posterior periosteum and parietal pleura. The Crosby-Cooney button is then placed in the pleural cavity selecting the length of the button that is deemed best suited for the case in hand. (The buttons come in lengths of ⅝″, ⅞″ and 1″.) The periosteum is then approximated around the flange in order to retain the button in place. The button is further anchored in place by using cotton sutures to approximate the external flange of the button to the intercostal muscle. The flap is reattached by means of a 2 layer closure. A continuous suture of No. 00 chromic is used for the deep fascia and interrupted and continuous silk is used for the skin." It is probably wise at the time of resecting the rib to also resect the nerve as the postoperative pain will be less.

CASE REPORTS

Mrs. G. had undergone an operation for carcinoma of the breast and subsequently developed a pleural effusion. Carcinoma cells were obtained from the pleural effusion. The effusion was reforming so rapidly that the attending physician found it necessary to tap her 3 times a week in order to prevent dyspnea and in order to keep the left pleural cavity partially evacuated. A Crosby-Cooney button was placed in the pleural cavity on May 3, 1955.

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She died approximately 6 months later. She was comfortable during this time and it was unnecessary to perform thoracentesis.

Mrs. T. also had a carcinoma of the breast with pleural metastasis. Repeated thoracentesis was necessary. A Crosby-Cooney button was placed in the pleural cavity on Jan. 16, 1956. She lived for 9 months without necessity for additional thoracentesis.

The third patient was a male, J. A. He developed pleural effusion, but the cause could not be determined. Papanicolaou cell study of the fluid failed to reveal carcinoma cells. A thoracotomy was performed. Histologic study was reported as revealing an adenocarcinoma with probable origin in the pancreas. A Crosby-Cooney button was placed and he remained comfortable without necessity for thoracentesis until his death 6 months later.

The fourth patient was a female, Mrs. W. N. The original diagnosis was carcinoma of the breast. A mastectomy had been performed. Bilateral pleural effusion was present which made her dyspneic. Repeated thoracentesis was necessary to keep her comfortable. A Crosby-Cooney button was placed first in the right pleural cavity in January 1956 and a second Crosby-Cooney button was placed in the left pleural cavity in May of 1956. These two buttons provided adequate decompression, without need for thoracentesis. The pleural effusion on the right reaccumulated in September of 1956 (8 months after insertion of the button) and at this time she was treated with nitrogen mustard (Sept. 9, 1956). An 8 mg. dose was placed in the right pleural cavity (diluted in 30 cc.) and, in addition, she was castrated. The nitrogen mustard caused her some pain, but no fever. This relieved her pleural effusion on the right side. The decompression of the left side remained satisfactory. It was concluded that the Crosby-Cooney button on the right had become obstructed so that good drainage was no longer present. The patient subsequently received another intrapleural injection of nitrogen mustard in January of 1957 and, at the present writing, (May 1957) (2 years following her first therapy for carcinomatous pleural effusion) she is able to continue her work as a secretary.

DISCUSSION

Carcinomatous pleural effusion is amenable to surgical therapy. The relief obtained by these patients is gratifying even though limited by the prognosis. The use of the Crosby-Cooney button is a simple procedure and it seems to be quite efficacious in most patients. Similar disappearance of the pleural effusion can be obtained by a pleurectomy. This allows for a large absorbing surface. It demands a major thoracotomy and in individuals who are in poor condition its performance may be questioned when simpler methods are available.

A word of caution is probably necessary with reference to the use of this button in bilateral pleural effusion due to congestive heart failure. In this type of case where the tissues are edematous, the intrathoracic fluid will not be absorbed into the chest wall tissues or at most the results will be disappointing in view of the water-logged condition of the tissues as the result of the congestive failure.

It seems pertinent to also mention other methods for treating carcinomatous pleural effusion, particularly with relationship to nitrogen mustard and colloidal gold. In a Cancer Seminar, in Phoenix, Arizona, Doctor Garland of San Francisco outlined his method of choice for treatment of carcinomatous pleural effusion. It was his contention that X-ray therapy will often dry up a pleural effusion and he believes that this should be given priority over any other method of therapy. His second choice was "simple drainage". By this term he more accurately meant thoracentesis as it was his experience that evacuation of the pleural effusion is followed by slow return of fluid or failure of the pleural effusion

to reaccumulate. His third choice was nitrogen mustard, but he did not seem too enthusiastic about this type of therapy. He was quite sure that he would *not* use colloidal gold. His experience, and the experience of other individuals interested in radio-isotopes, have indicated to them that the use of colloidal gold is fraught with hazards and has nothing to offer that cannot be accomplished in just as satisfactory a manner with other means. Some individuals have found "phosphorous 32" to offer all the advantages and none of the disadvantages (i.e. secondary irradiation) of colloidal gold.

A further word might be said with regard to nitrogen mustard and its use in intrathoracic malignancies. Doctor H. B. Eisenstadt of Fort Arthur, Texas, has pointed out that although the majority of patients who undergo treatment with nitrogen mustard are benefited by it, a few may show adverse reactions which are similar to those at times encountered after X-ray therapy or other methods of irradiation of the lung. Among other untoward reactions, he reported 3 cases which closely resembled acute irradiation pneumonitis.

SUMMARY

Surgical decompression of carcinomatous pleural effusion can be accomplished by use of the Crosby-Cooney button. Four cases are described herein in which the patients were successfully treated by the use of the button. The relief of the pleural effusion has given the patient symptomatic relief and repeated thoracenteses have been obviated. Other methods for treating carcinomatous pleural effusions are mentioned and remarks made with regards to their efficacy. The Crosby-Cooney button should not be used for decompression of pleural effusion based on congestive heart failure.

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POSTOPERATIVE CARE: THE ROLE OF THE RECOVERY ROOM

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The morbidity and mortality of the operative patient is increased in the immediate postoperative period. Prior to the establishment of the Recovery Room, or Post Anesthesia Recovery Unit (PAR), as it is properly called, the lack of trained personnel for the constant supervision of the postoperative patient posed a great problem both from the economy of time and personnel as well as equipment. Untrained personnel were given the job of staying with the patient until his emergence from the anesthetic, and this occasionally led to many accidents not immediately recognized. By the establishment of a PAR Unit this mortality and morbidity has decreased. A high caliber of preoperative preparation, safe and competently administered anesthetics, and skilled surgery, can be negated by haphazard immediate postoperative care.[1] Many postoperative complications such as shock, cardiac arrhythymias, and anoxia are handled efficiently by the PAR Unit personnel. Proper management and care of the patient by well trained and experienced personnel is essential in maintaining the efficiency of the PAR Unit.

Hospitals can utilize space in or near the surgical suite, and convert it into a functioning PAR Unit. The size of the unit depends on the requirements of that institution, but the basic set-up together with the principles of management and care are fundamentally the same. Hospitals under construction or in the planning stage include in their plans adequate facilities for a PAR Unit. Established hospitals can convert space into a PAR Unit allowing one or two beds for each operating room of the surgical suite. Each recovery bed should have adequate working space surrounding it, approximately 8 by 6 feet.[4] Ideally the PAR Unit should be located away from the main traffic of the hospital, although with easy access to the operating suite. The transportation of an operative patient from an air-conditioned operative suite should necessitate having the PAR Unit likewise air-conditioned, so that the heat regulatory mechanism of the patient is not affected.

Prior to its use as a PAR Unit, the physical plant at St Joseph's Hospital, Wichita, Kansas, functioned as a play and sun room for the patients. Being ideally located directly across from the operating suite, it was considered to be the ideal location for such a unit.

Essential equipment necessary to care for any postoperative patient is centralized in the PAR Unit. The following equipment is listed as a guide:

1. Beds or carts with side rails. The carts should be of the type which can be placed in Trendelenberg position.

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2. Oxygen Equipment. This may be of the fixed or portable type. It should be individual with each bed or cart present.

3. Suction Equipment. Regular suction equipment of the fixed or portable variety may be used. It may or may not be for individual use.

4. Inhalation Equipment. This may be for therapy or resuscitation. Use of the Bennett Positive Pressure Breathing therapy unit is recommended.

5. Records. These may be separate sheets, or may be on the reverse side of the operative record.

6. Nurses' Station.

7. Telephone

8. Cabinet for drugs. There should be a locked space for narcotics.

9. Storage space for fluids. A variety of parenteral fluids are stored together with the sterile sets for their administration.

10. Drug tray. This should contain the necessary vasopressors and stimulants.

11. Air-conditioning.

12. Sterile instrument pack containing sterile cardiac resuscitation equipment. This must be complete and accessible.

The physical organization of the PAR Unit is individual for each institution, but should be so planned that a few well trained personnel can care for the patients without too much added effort. One graduate nurse with special training should be available for each two beds, although one for each bed is the ideal situation. Student nurses should take part of their surgical training in the care of the postoperative patient. In addition, one or two orderlies should be available to transport the patients back to their hospital rooms.

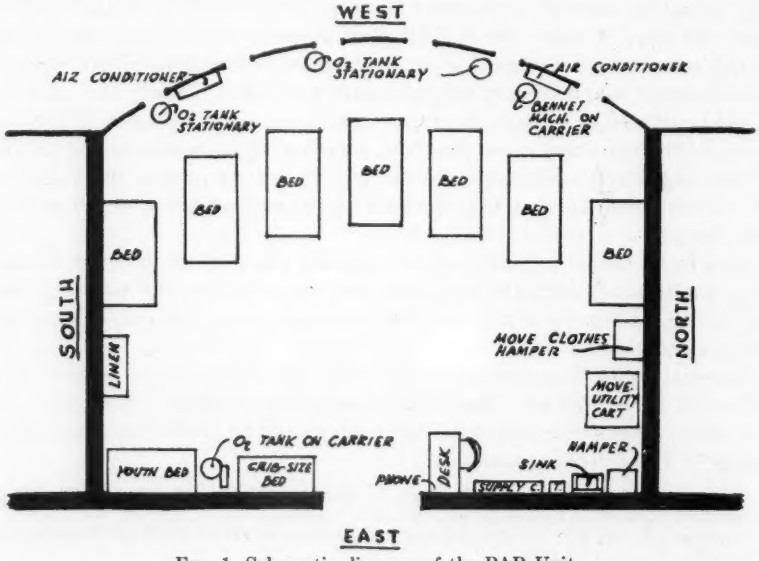

FIG. 1. Schematic diagram of the PAR Unit

FIG. 2. The functioning PAR Unit

The care of the patient begins with his entrance into the PAR Unit accompanied by the anesthesiologist. He is postured according to his needs, but is usually placed in the lateral Sims position with the uppermost leg flexed and the head extended. A pillow is placed both against his back and abdomen to keep him in position. Oxygen is started by means of a nasal catheter, taking care to place the catheter correctly. With the use of a tongue blade, depress the tongue and visualize the oropharynx. Insert the catheter into the nose, and push it forward until the tip of the catheter can be seen at the uvula. The catheter is then fixed in place by the use of adhesive tape. Levine tubes, thoracic and urinary catheters are attached to their proper bottles. The pulse, blood pressure, and respirations are checked and charted, and a record kept every 5 minutes until the emergence from the anesthetic has progressed sufficiently so that the patient knows his name, address, date, operation performed, and physician's name. He is then considered to be capable of returning to his room without further watching.

The care of the patient is usually centered around (1) the respiratory system, (2) the circulatory system, (3) pain, (4) the gastrointestinal system, (5) prevention of injury to the patient.

Respiratory depression is the most commonly encountered respiratory complication. It may be due to an overdose of the anesthetic agents, or drugs. If apnea is present, prompt resuscitation with oxygen administered by a bag and mask is the method of choice. This should be administered until the respiratory

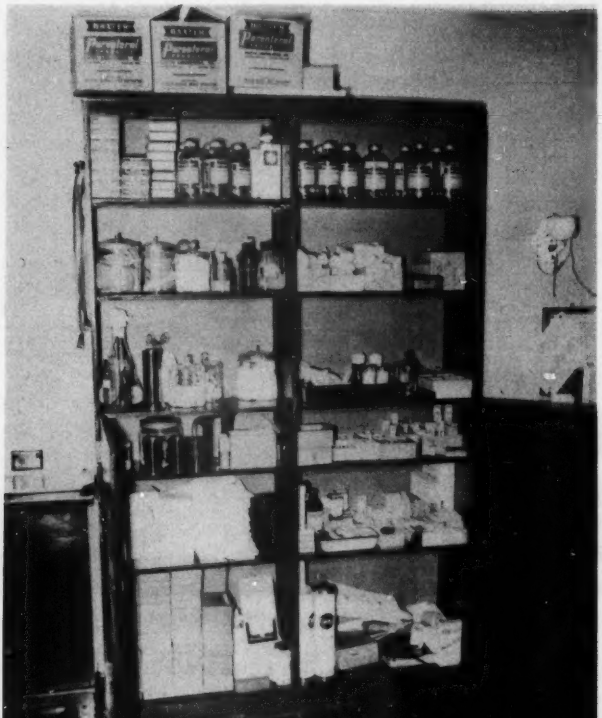

FIG. 3. Storage cabinet for fluids, drugs, and necessary equipment

movements are normal. A clear airway should be maintained by a metal or plastic oral airways, or by the insertion of an endotracheal tube. This should remain in place until its needs are terminated. Depression due to drugs should likewise be treated first with the administration of oxygen. Injection of 'Lorfan' and 'Nalline' terminates the depression caused by narcotics, and is usually effective. 'Nalline' is short acting, and may have to be repeated in the period of an hour, while 'Lorfan' is a much longer acting drug, and may have its effects last as long as 3 to 4 hours. The use of Coramine and other analeptics is of no value in restoring normal respiratory rates, and should not be used in a PAR Unit. Atropine sulfate should be used in the presence of laryngospasm and bronchospasm. However the most important adjunct to normal recovery is the administration of oxygen. Pulmonary edema, when present as a respiratory complication, is treated by the elimination of secretions, if possible through an endotracheal catheter, or otherwise through a tracheotomy. Positive pressure inhalations of oxygen helium mixtures, and ethyl alcohol vaporizations to reduce

the surface tension of the secretions are also used to relieve the edema. Atelectasis, massive collapse, tension pneumothorax, and hemothorax are diagnosed promptly by clinical means, and many patients are routinely given the benefit of the portable roentgenograms. Bronchoscopy and oxygen therapy are promptly performed as indicated.

Shock is probably the most commonly encountered of all the postoperative complications. Shock has to be differentiated from the hypotension which occurs frequently with the use of spinal anesthesia. The latter may present a systolic pressure below 80 mm., however the pulse is slow and steady.

Shock is characterized not only by a drop in the systolic pressure below 80 mm., but also by a fast, thready pulse, pallor, sweating, and changes in the character of the respirations. Early diagnosis of shock is extremely important to the welfare of the patient. The reversal of the peripheral circulatory collapse to its normal state can be accomplished by the immediate recognition of the cause. The patient should be checked for hemorrhage at the operative site. The movement of the patient from the operating table to the cart can at times cause the patient to go into shock. The exact mechanism of this condition has not been determined, although it has been investigated.[3] Fluids by the parenteral route should be started unless the patient has returned from surgery with intravenous fluids running. The severity of the operation is not an indication of which patient may be a candidate for shock. Many minor procedures result in trauma to the patient with resultant shock. Blood, plasma expanders, vasopressors, and cortisone are all used in the treatment of shock. Blood banks in the hospital afford necessary blood replacement with minimal effort. Plasma expanders such as Gelatin, Dextran, and Oxypolygelatin are used to restore the circulating volume and maintain the blood pressure until blood replacement can be made available to the patient. Since shock constitutes peripheral vascular collapse, the use of vasopressors to combat this collapse is applicable. Epinephrine, as well as ephedrine, should not be used in patients in whom the shock is due to peripheral vascular collapse. These drugs act on the myocardium directly, accelerate the heart, and increase the work load. Drugs, such as 'Vasoxyl' and 'Neosynephrine', cause increased peripheral resistance without evidence of direct cardiac stimulation[2]. It may be advantageous in the treatment of shock to inject 15 mg. of 'Vasoxyl' intramuscularly when the pressure has fallen below 90 mm. systolic pressure. If this does not raise the pressure within 10 to 15 minutes, the use of 'Vasoxyl' by the continuous intravenous infusion may be necessary. Two hundred fifty cc. of 5 per cent dextrose containing 35 to 40 mg. of 'Vasoxyl' is suggested. The rate of flow is altered to the response of the blood pressure. However, when hypotension is associated with hemorrhage, the usual methods for restoring blood volume are required, even when 'Vasoxyl' is used to restore normal vascular tone. The use of 'Levophed', norepinephrine, in dilutions of 4 cc. to 1000 cc. 5 per cent dextrose, by intravenous infusion is also effective, and may be used over a long period of time. The administration of oxygen, even though the respiratory exchange seems adequate, relieves the hypoxia which is always present, because of the reduction of the circulating blood volume. The adminis-

tration of 'Solu-Cortef', 100 mg. in 2 cc. diluent has at times given miraculous recovery from shock. However it should not be given as the only treatment of shock, but as one of the many methods of therapy for shock.

Cardiac arrest, although relatively uncommon as a circulatory complication in the PAR Unit, may be successfully treated in the PAR Unit. Success depends on instant detection and therapy. The left chest should be opened immediately, and the heart massaged, while assistants insert an endotracheal tube and establish artificial respiration. Defibrillation, injection of adrenalin and calcium chloride may be required to establish continuity of the heart action.

The restlessness of the patient following recovery from the anesthetic should not be interpreted as a response to pain by the patient. Pain causes limitation of movement and depression of respiration. The patient should be checked for abnormal position, tight dressings, distention of the bladder, and distention of the abdomen. Correction of these may reduce the restlessness of the patient. The causes of restlessness are pain, hypoxia, and mental depression. These result from the premedication or the anesthesia. The administration of narcotics postoperatively should be in small quantities, because of the depressant effects of the drugs, and because there still remains in the blood stream some circulating anesthetic agents. There should not be any routine administration of narcotics when the patient first reacts from his anesthetic. His repositioning and checking for any unnecessary external stimuli should substitute for the administration of a narcotic at that time. If a narcotic is necessary, then Demerol should be the one of choice, as it is a mild analgesic with a better safety factor than morphine. The patient has to be watched carefully during the reactive period, and the side rails on the bed or cart must be in place. Restlessness can be corrected by the use of oxygen and by the discriminate use of sedation. Oversedation in the immediate postoperative period can be the cause of prolonging recovery periods.

The most frequent gastrointestinal complication is nausea and vomiting. With the use of a series of drugs, such as 'Thorazine', Dramamine, and Marezine, the incidence and severity has decreased. Aspiration of vomitus should be treated immediately with the use of suction. It is possible to pass a catheter through the nose, and by proper extension of the head, have the catheter pass through the vocal chords into the trachea. Irritation of the trachea results in spasmodic coughing, and most of the aspirated material can be sucked into the catheter. Distention of the stomach can be relieved by the proper insertion of a Levine tube.

Records are essential in the PAR Unit. Detailed charting by the nurse on the reverse side of the operative record or by the use of a separate PAR Unit record, gives the surgeon and anesthesiologist an immediate indication of the status of the patient. All information charted on the record following the patient's entrance into the PAR Unit becomes a part of the permanent record, blood pressure, pulse, respiration, fluids, narcotics, and activity of the patient in the PAR Unit is easily visualized on the record. The record should be reviewed by the attending physician or the anesthesiologist before the patient is returned to his room. Daily statistics are also kept, and tend to give a summary of the activity

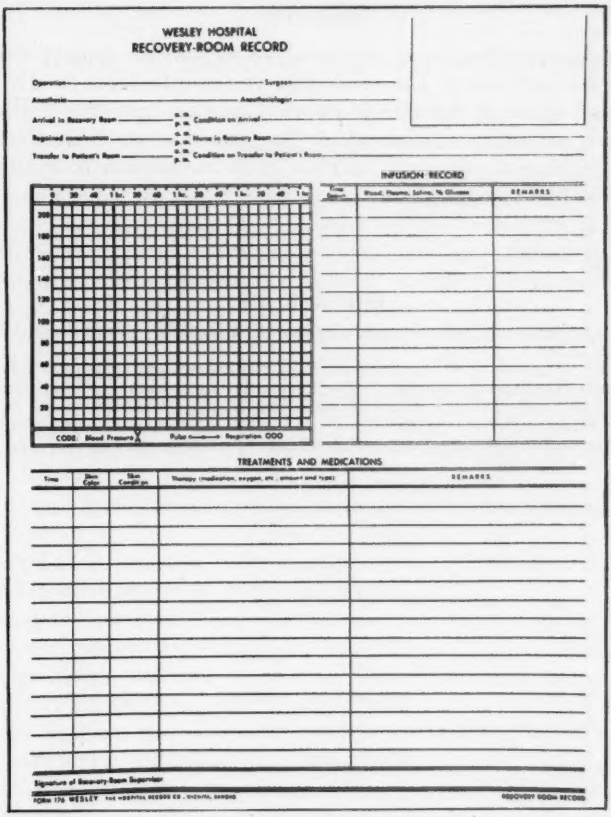

FIG. 4. Recovery Room Record

of the PAR Unit on a daily, monthly, or yearly basis. During the past 2 years, a total of 6540 surgical patients have been treated in the PAR Unit of St. Joseph's Hospital. This includes out-patients who were given general anesthesia as well as those who were hospitalized. The average stay per patient was 1 hour and 21 minutes.

There are a few disadvantages to the operation of the PAR Unit. Some feel that there is a lack of privacy for the patient. Male and female patients are placed next to each other, and it is thought that the patients might object when they have reacted sufficiently. We have noticed no objection to the lack of privacy. With proper explanation to the relatives concerning the function of the PAR Unit, there has been no objection on their part, and we have received excellent cooperation from them. The only real disadvantage to the PAR Unit is that it causes a reduplication of beds. This, however, is a minor disadvantage when weighed against the advantages to the patient in terms of expert care and safety.

SUMMARY

The need for centralization of postoperative patients, equipment, and personnel to care for their needs, has resulted in the establishment of a PAR Unit. The increased safety in the care of patients has been reflected in the reduced postoperative morbidity and mortality. The success of the PAR Unit depends on proper training of personnel, and the early recognition of anesthetic and operative recovery complications.

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SURGICAL REPAIR OF RADIATION INJURIES

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Advances in radiation research are everyday news. Twenty million, even 500 million volt x-ray or gamma ray machines are in the making. Experimental work with electron, proton, neutron, and deuteron beam therapy is in progress. Nuclear weapons and power plants with their known and unknown hazards are here—now.

All of these in addition to our old friends, radium and x-ray, whose occasional deleterious effects we still seek to control.

To be sure, in comparison with the huge number of treatments given with roentgen rays and radium, the actual total number of injuries is relatively small. To the plastic surgeon who sees these patients, however, the number seems large.

A grouping of patients suffering the ill effects of radiation has been given by Cronin.[4]

Group I: Lesions resulting from a single massive dose, intentional or otherwise; or a few gross exposures at short intervals.

a. Prolonged fluoroscopy.
b. Improper dosage or other technical error.
c. Atomic energy workers.
d. Excessive dosage in treatment of malignancy.

Group II: Patients receiving many exposures over a long period of time.

a. In treatment of various benign conditions.
b. Physicians, dentists, technicians.
c. "Shoppers".

Group III: Patients who have received well directed irradiation by competent radiologists for malignancy or other conditions, who, after many years, develop the usual irreversible changes in skin and underlying tissues.

The delay in tissue response to radiation is well-known. Hempelmann[8] believes the subtlety of the changes in intracellular mechanisms; e.g. enzyme systems, is responsible for this.

The characteristic histologic picture of radiation injury was outlined by Wolbach[12] in 1925 and has been re-emphasized by Brown[1] and by Mason.[10] Greeley[7] summarizes these changes as follows:

Atrophy of the skin and its appendages, telangiectasis, arteriolar and venous sclerosis, fibrosis, avascularity with irreversible obliteration of arterioles, venules, capillaries, and lymphatics, chronic degeneration and ulceration, and finally carcinomatous degeneration.

The above changes are progressive.

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The hazards and problems of surgery in these patients have been listed by Robinson[11] as infection, slough, exposure of vital structures (tendon, bone, great vessels, nerves, brain), difficult hemostasis, difficult dissection, radioosteonecrosis, poor demarcation of normal and devitalized tissues, general inanition, pain, and narcotic addiction. Of these, the latent period in the development of slough has assumed new importance since with newer radiation modalities, the deep tissue changes may be delayed for months or even years. In addition, overlying skin and soft tissues may appear relatively normal, yet obscure deep fibrosis or necrosis.

Conway[3] succinctly states the plastic surgeon's dilemma "as one of wound healing to be accomplished in tissues known to be deprived of their normal capacity for growth and repair".

Preoperatively, these patients require careful planning for reconstruction and precise evaluation of their general condition. Control of infection, surgical drainage, removal of sloughs and sequestrae, restoration of nutritional status, and correction of existing anemia are of the utmost importance.[2]

Despite the hazards, there is general agreement that the treatment of radiation injuries is surgical. The principles upon which successful reconstruction must be based are quite well established.[5, 6, 9] Surgery is contraindicated during the acute stage of reaction. Wide surgical excision of the involved tissues is imperative. Immediate surgical repair should be done whenever possible, lest further slough from the open wound occur. Repair should be accomplished with local flaps carrying their own blood supply whenever possible. Distant flaps delayed or otherwise, should be employed only when the aforementioned technics are out of the question.

SUMMARY

The hurried pace of nuclear energy research accents the timeliness of any discussion of radiation injuries. Tissue changes caused by excessive radiation are not confined to ulceration or malignancy, but affect surrounding tissues to a variable degree. The extent of injury is difficult to determine since with newer modalities of radiation, the latent period assumes greater importance.

Treatment of radiation injuries is surgical. Wide excision with immediate reconstruction should be employed whenever possible. Surgery other than minimal debridement is contraindicated during the acute phase. The chronic radiation changes are progressive with a decided tendency for malignancy to develop often after many years.

One should be familiar with the hazards incident to this type of reconstructive work, yet be cognizant of the fact that often these painful ulcers and malignancies can be treated by adequate surgery with restoration of these patients to useful life.

Like most injuries, the vast majority can be prevented.

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INCISIONAL HERNIA

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The abdominal wall repairs itself by scar tissue, a fact well known for many centuries. Even before the Christian Era, hernias of the abdominal wall were treated by cautery and caustics. The method produced excessive scar tissue and in many instances cured the hernia.

Following some of our surgical incisions, the layers of the abdominal wall failed to heal properly for one reason or another, and ventral or incisional hernias occur. At times they become very large in size and, consequently, are difficult to repair surgically.

We will all agree the best surgical method of repair is by excision of the old scar, excision of the sac and layer to layer repair without tension on the suture line. How often can this be done in large hernias? To get the muscular and fascial layers together without tension is rarely possible. Masterful work has been done by Koontz and others on repairing these defects with fascia, tantalum mesh, or fortisan fabric. The use of these materials is most helpful in the repair of some of the large hernias as many of them cannot be repaired without employing some foreign material to bridge the defect. When this condition occurs these materials should be used in their proper manner.

Frequently in cases of incisional hernia there is present a good strong sheath of scar or fibrous tissue which can be used in the repair of these hernias. This has been demonstrated and reported by many authors on the subject, notably, Babcock, Cattell, Koontz, and others. It is my belief that this principle is often overlooked by surgeons. Many times the strong tissue is cut away and discarded at the very beginning of the operative procedure. The principle, although often described is not, in my opinion, taught or utilized sufficiently. We hopefully excise the sac and attempt to do a nice artistic layer to layer closure only to find a large defect left which cannot be closed except with great tension on the suture lines, with a resulting high recurrence rate.

Babcock in 1925 described interdigitation in the repair of large ventral hernias. To quote, "Frequently the sac is quite thick and strong, and if sacrificed, tissue is lost that might give added support, and that compares well with a transplant of fascia lata." Figure 1 illustrates his method.

Cattell in 1942 described a technic for the repair of incisional hernia. To quote, "This layer dissection may be combined with an overlap method whereby the fascia is imbricated to form a double layer at the point of greatest weakness." Figure 2 illustrates his method.

Koontz in many of his papers on this subject refers to the fact that tissue present, which may be useful in the repair of the hernia, should not be discarded.

Presented during the St. Petersburg Assembly of The Southeastern Surgical Congress, April 1–4, 1957.

From the Johnston-Willis Hospital, Richmond, Virginia.

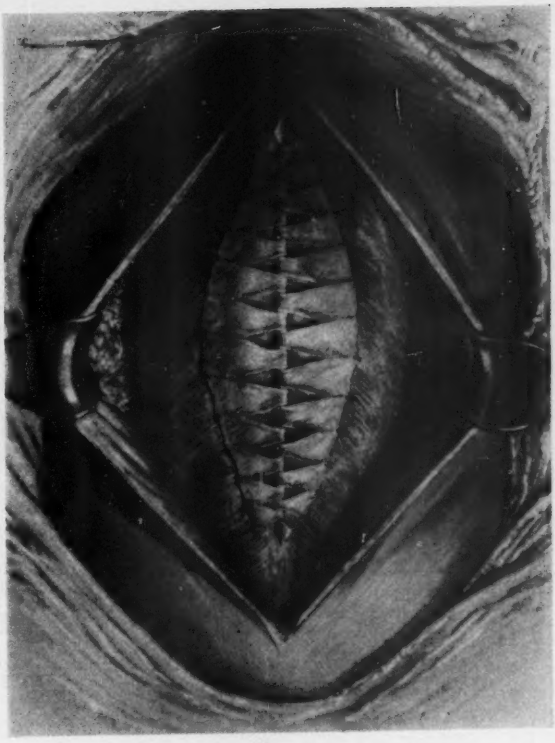

FIG. 1

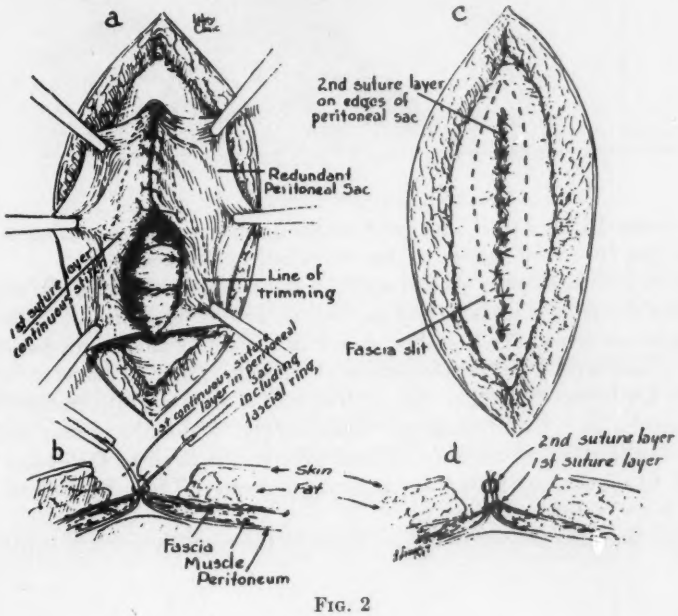
a
c
2nd suture layer on edges of peritoneal sac
Redundant Peritoneal Sac
Line of trimming
1st suture layer continuous stitch
1st continuous suture layer in peritoneal sac including fascial ring
Fascia slit
b
d
Skin
Fat
2nd suture layer
1st suture layer
Fascia
Muscle
Peritoneum

FIG. 2

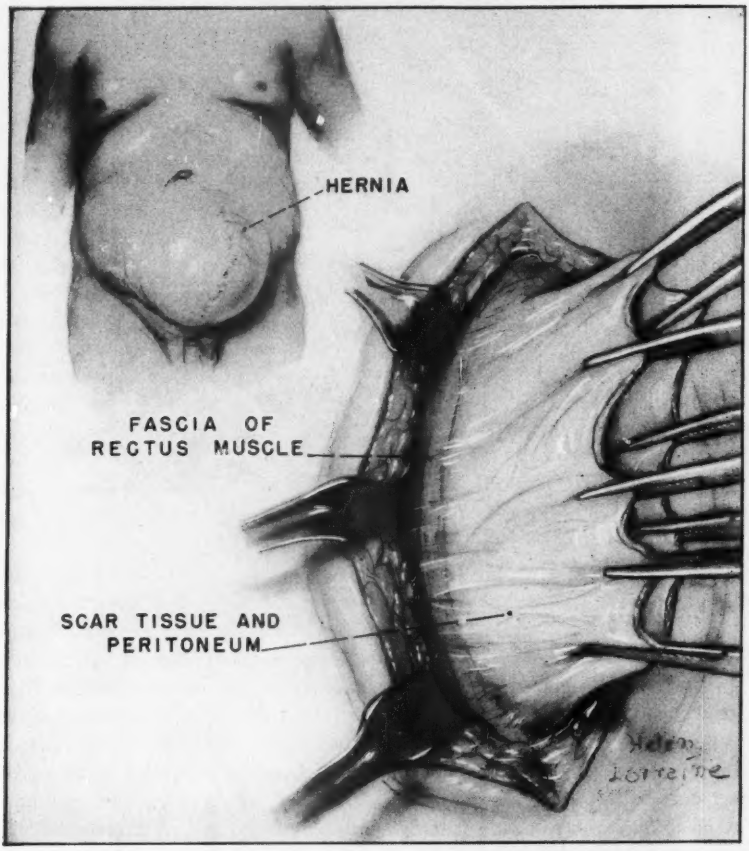

FIG. 3

The method I have used with good results in several patients during the past 5 years has been very similar to that described by Babcock.

The old skin incision is excised and the skin is separated from the scar and the dense sheath of scar tissue and sac (fig. 3). The sac is opened in the midline. Adhesions are freed. The posterior sheath of the rectus along with peritoneum is first closed with a continuous suture of chromic catgut. If possible the recti muscles are brought together (fig. 4). The scar is then cut into ribbons with a wide base so as not to damage the blood supply. The strips of scar with scar tissue sheath attached are then crossed over and sutured to the rectus fascia (figs. 5, 6). Drains are used when it seems necessary. The subcutaneous tissue and skin are closed.

I have used this procedure several times in the past 5 years and believe that

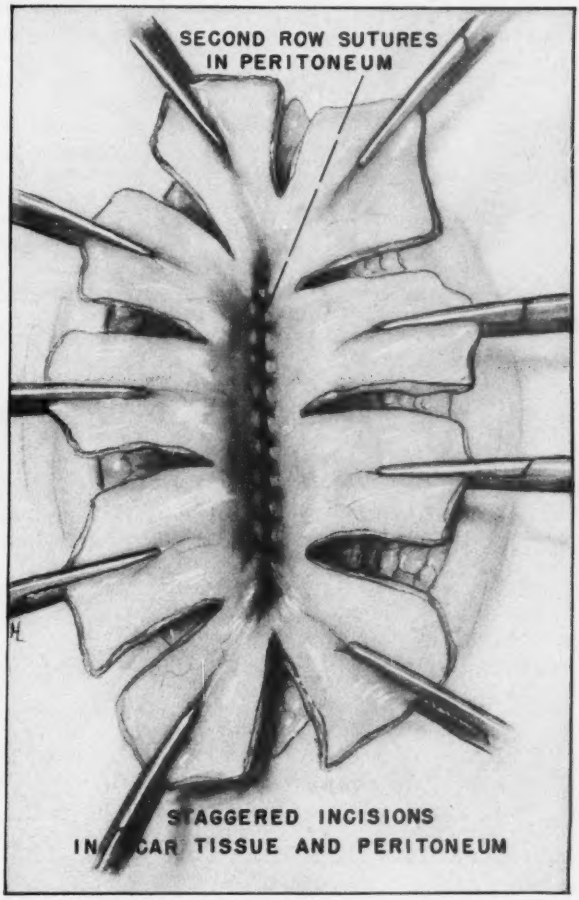

FIG. 4

it is most successful in the repair of certain cases of incisional hernia. It gives a strong support to the abdominal wall. All incisional hernias cannot be repaired by this method, but it is a useful adjunct.

Those with very large defects can be repaired by using this technic and further reinforcing the closure with some material such as tantalum mesh or fortisan fabric (fig. 7). The small incisional hernias can be repaired by the classical technic of excision of the scar tissue and repair of the hernia in layers.

CASE REPORTS

Case 1. A white man, aged 51, had 7 years previously had an upper midline incision for surgery of an ulcer. He did not know just what type of operative procedure had been per-

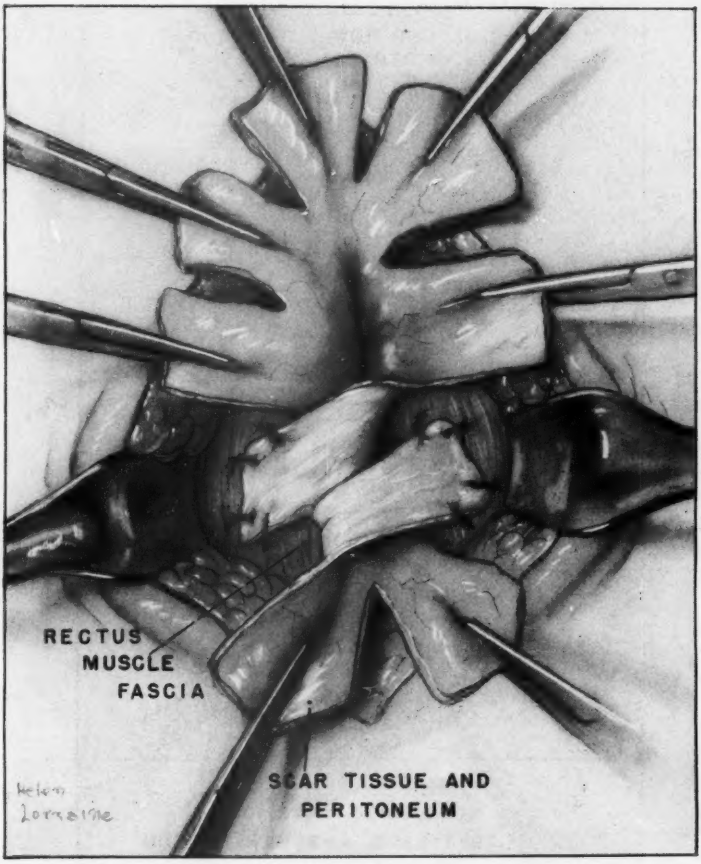

FIG. 5

formed on his stomach He had a large incisional hernia which was described by the intern as being the size of a gallon can. The skin over the area was ulcerated. The hernia was repaired in February 1953 using the technic discussed above. After completing the repair, a piece of tantalum mesh was used and sutured to the fascia with interrupted sutures of silk.

He has remained well.

Case 2. An obese white woman, weighing 250 pounds, aged 47, was operated on by the author in August 1953 for a large ovarian cyst through a low midline incision. Postoperatively she developed abdominal distension, and a large incisional hernia occurred. This gradually became larger and more painful. In February 1954 it was repaired by the described method. She has remained well in spite of the fact that instead of losing weight as she has been advised, she continues to gain.

Case 3. A 60 year old white woman had elsewhere undergone an abdominoperineal resection of the rectum for carcinoma. The colostomy had been brought out through the low midline incision. A very large incisional hernia was present. The colostomy was transferred

to the left lower quadrant of the abdominal wall. No evidence of recurrence of the tumor could be felt within the abdominal cavity. The same procedure was followed in the repair of this hernia in January 1953.

There was no evidence of recurrence of the hernia at the time of her death from metastatic disease about 2 years later.

Case 4. A 52 year old obese white woman complained of low abdominal pain. She had previously had pelvic surgery; a supracervical hysterectomy having been performed. A large incisional hernia was present at the site of the low midline scar. After the adhesions were freed, a mass could be felt in her pelvis. This was removed and the pathology was that of leiomyoma. In July 1955, the hernia was repaired with the same technic. Silk was used in the closure. A small abscess developed which was opened and silk sutures removed.

The closure has remained intact.

Case 5. An obese, severely arthritic, white man, aged 60, had an abdominoperineal re section of the rectum in March 1954. No evidence of metastatic disease was found and the regional lymph nodes were not involved. A low midline incision was made and the colostomy was brought out through the incision. The patient developed distension postoperatively

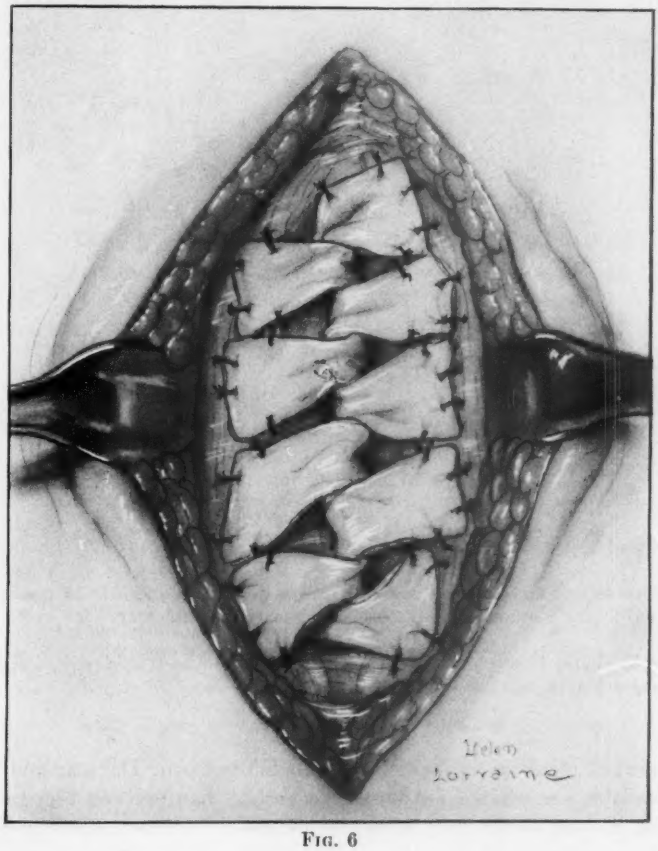

FIG. 6

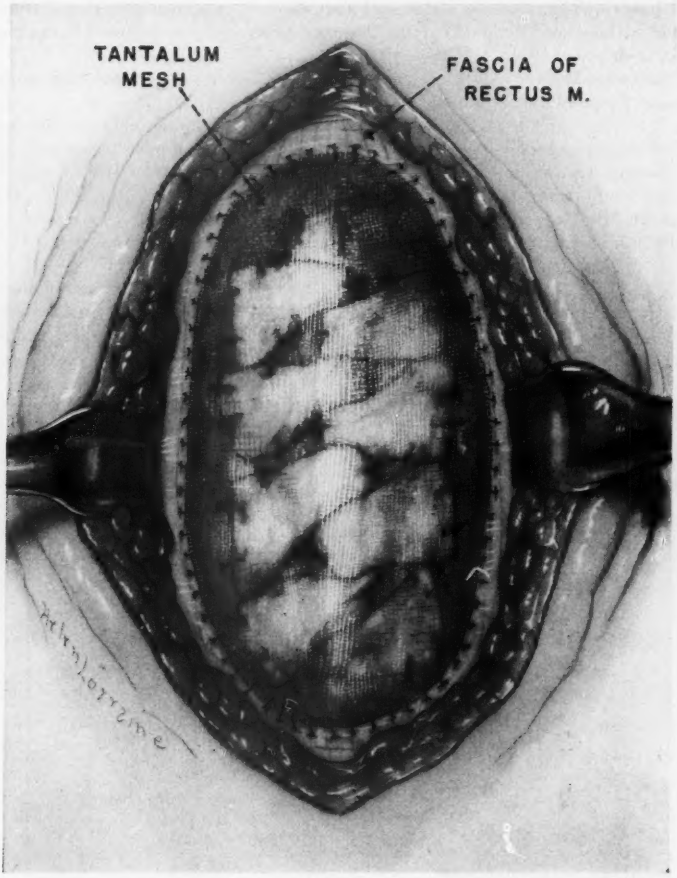

FIG. 7

and the wound became infected. A large incisional hernia developed and continued to enlarge.

In October 1955 the colostomy was transferred to the left lower quadrant and the herna was repaired by the method under discussion. Metastatic nodules were felt in the liver at this time.

He died in August 1956 from metastatic disease, and post-mortem examination revealed the repair was holding.

SUMMARY

A method of repair of an incisional hernia is discussed. The scar and fibrous tissue available are utilized for the repair rather than excising the tissue and discarding it. The defect can be reinforced under tantalum mesh or fortisan

fabric if desired. The method has been described before, but I beilieve it is too infrequently used.

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EDITORIAL

POOLING OF ANCILLARY SURGICAL FACILITIES

The need for additional special diagnostic and therapeutic facilities resulting from the incorporation into clinical practice of the many new developments in physics, chemistry and the biologic sciences is being increasingly felt in all branches of medicine. In no field is it being encountered more than in clinical surgery.

Diagnosis, preoperative evaluation and preparation as well as treatment, including operative management and postoperative care, are requiring more and more elaborate equipment and highly trained ancillary personnel. The complete fulfillment of this need requires the securing and housing of much expensive equipment which rapidly becomes obsolete or which for other reasons, must be frequently replaced at ever-increasing costs. In many instances, such equipment remains idle much of the time when its availability is limited to a single institution.

Just as important as is the acquisition, housing and maintenance of such equipment is the procurement of the specially trained personnel to use it or to provide other services which, although not requiring expensive equipment, demand special knowledge or technical ability. Hospitals and medical schools are finding themselves in increasingly disadvantageous competition with industry and other organizations for the limited supply of chemists and physicists, as well as other basic scientists and technicians. Ironically, in some communities there is unnecessary duplication of certain equipment and personnel, while at the same time there is a shortage or a complete lack of other facilities.

In all but a few well endowed institutions it is already being found impossible to meet the ideal requirements, with the result that in many communities considerable deficiencies now exist. In the face of constantly increasing basic service, maintenance and housekeeping costs, and with charges to patients having already reached such a high level, there is no reason to expect any improvement in the foreseeable future, unless cooperative arrangements can be made between the medical institutions in each community or larger geographic areas, as the case may be.

Instead of each hospital in a community futilely attempting to secure and house all the equipment and to procure and adequately pay the qualified ancillary personnel, hospitals in most communities could come nearer to solving their problem by pooling arrangements with other hospitals and with medical schools, thereby providing more adequate facilities for each of the participating institutions. Without such cooperative arrangements, most communities will find it impossible to offer medical and surgical services which meet the highest present standards. That such a plan is practical has already been amply demonstrated in many communities where it has been tried.

The method of coordination and the precise mechanism, including the financ-

ing of the plan, the acquisition of the equipment, and the procurement of personnel for accomplishing such a pooling endeavor must necessarily vary, depending on circumstances in each community or area. In some instances, already existing buildings might be used, while in other instances new buildings physically apart from any of the participating institutions might be desirable. Except in the instance of fixed equipment which requires the presence of the patient for diagnostic or therapeutic procedures, the patient would not necessarily have to go to several hospitals or be transported from one place to another. When only examination of a specimen is involved, that could be sent from one institution to another by hospital attendants. In the instance of a least certain portable equipment, it along with the required personnel might be transported from place to place as needed.

As in all undertakings of this sort, its development and successful implementation is primarily dependent on education of everyone concerned, as it is imperative that there be widespread conviction of the need, merit and feasibility of the plan. Those whose understanding and trust must especially be gained include clinical practitioners, medical school faculty members and administrators, hospital administrators and architects, and the general public. The persons responsible for the planning of future community medical services must be made aware of the expectable ever-increasing need to provide for pooling of special facilities.

Such a venture is likely to be fraught with many difficulties. The desire of many hospital and medical school administrators and faculty members to keep their institutions completely autonomous or to have them appear to advantage over other institutions, as well as certain expectable distrusts, petty jealousies and myriad other obstacles, including passive resistance, misunderstanding and apathy, are obstacles which must be anticipated, dealt with, and overcome. Honesty, patience, frankness, and mutual understanding and trust are essential requisites if such a plan is to work.

Because it concerns them so much in their daily practice, and because they are frequently in the best position to influence community thought in matters of this sort, practicing surgeons in both the younger and the elder-statesmen groups must take the lead in promoting the pooling of special ancillary hospital facilities.

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BOOK REVIEWS

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

Peripheral Nerve Regeneration. A Follow-up Study of 3,656 World War II Injuries. VA Medical Monograph. By BARNES WOODHALL, M. D., AND GILBERT W. BEEBE, Ph.D., U. S. Government Printing Office, Publishers.

Without any thought of relegating the scientific data and the investigators' conclusions to a secondary position, it is necessary to focus attention upon the invaluable contribution this monograph offers in the administration of a mass clinical follow-up. All physicians interested in clinical research can profit by the experiences of these investigators in gaining patient participation, case sampling, standardization of testing by large numbers of clinicians, and the adopting of automation to the accumulation and computation of data.

Any criticism directed at this study because of mode of injury or its restriction to the veteran population, is more apparent than real. Our civilization, in peace as in war, produces similar injuries which confront most physicians. From this study, each physician can better evaluate seriousness of associated soft tissue, bone, joint, and vascular injuries, and establish the prognosis more accurately for each nerve and site of injury.

JACK SHARRETT, M.D.

Trophoblastic Growths—Hydatidiform Mole and Chorionepithelioma. By J. SMALBRAAK. Elsevier Publishing Company, Amsterdam, 1957. Distributed by D. Van Nostrand Company.

This monograph is a very complete work covering the world's experience and thinking in regard to hydatidiform mole and chorionepithelioma. It includes a large series of cases collected by the author with excellent analysis and follow-up. All significant aspects of trophoblastic growths are treated in a comprehensive, yet understandable manner.

The book is divided into two parts for separate consideration of hydatidiform mole and chorionepithelioma. The organization of material effectively clarifies the difficult, and often, confusing subjects.

There is an extensive analysis and comparison of the results and divergent views in the world literature including the author's series. The subject is discussed with perception and conclusions made by the author are logical.

The excellent chapter on histopathology is illustrated with outstanding photomicrographic plates. Diagnostic features including hysterosalpingography, hormonal studies, roentgenographic studies, cystoscopy, and pathology are evaluated carefully. The analysis of therapeutic results, including surgery, radiation, hormonal therapy, nitrogen mustard, and immunization procedures leads to interesting and provocative conclusions.

This book is a significant contribution in an area where, in spite of extensive literature, there is a great lack of organization, summarization, and unanimity of thought.

EDMUND B. MIDDLETON, M.D.

Operative Surgery, Vol. 3. By: CHARLES ROB AND RODNEY SMITH, Butterworth and Co., London.

This volume of the operative surgery series prepared by Drs. Rob and Smith maintains the excellent coverage and presentation of subject matter as was observed in the first two volumes.

The step by step presentation, with excellent text and illustrations, continues to be an outstanding feature of this book.

The first section of this volume is a continuation of volume two and completes the discussion of the large bowel. The majority of this volume is devoted to the thorax.

It is regretable that the authors did not complete the section on the large bowel in the previous volume and allow this volume to be devoted solely to the thorax as there are those who might be only interested in one of these two subjects. This, however, does not detrate from the overall desirability of this book.

ROGER D. SCOTT, M.D.

The Nurse and the Out-Patient Department. BY A. WINDEMUTH, The MacMillan Company, New York, 1957.

This textbook for nurses engaged in out-patient clinics, represents an effort to set forth theory and practice in an area where the scope is increasing steadily.

The role of the out-patient nurse in health education receives detailed treatment, and this is, indeed, a very important phase of her work. The initial chapter on the history and development of out-patient work is of interest to anyone connected with a hospital where it has out-patient facilities.

The reviewer questions the inclusion of some detailed material in this book, such as special diets appropriate for various disease conditions, on the ground that this material is easily available elsewhere. On page 438, there begins a section on what an infant would say if he could talk. It begins with "I am I", and ends 11 pages later with ". . . I can do a lot of things!".

On the whole, this text is bulky and frequently dull.

GEORGE ENTWISLE, M.D.

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Book Reviews and Editorials are grouped together and are indexed under those headings in alphabetic order.

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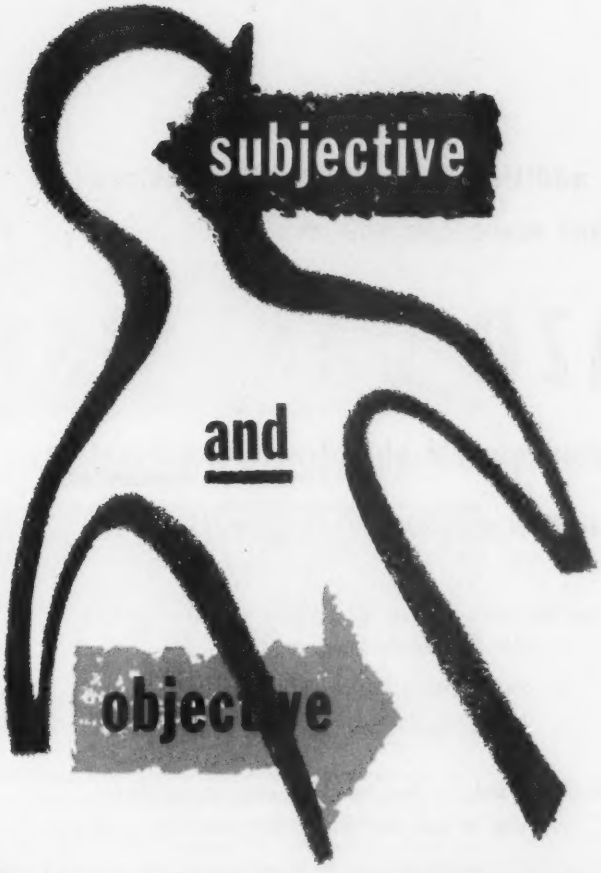

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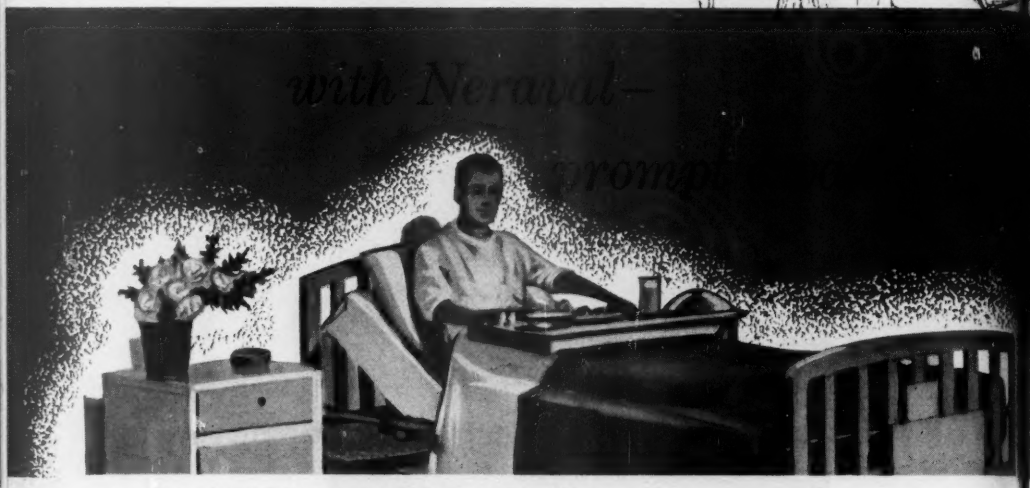

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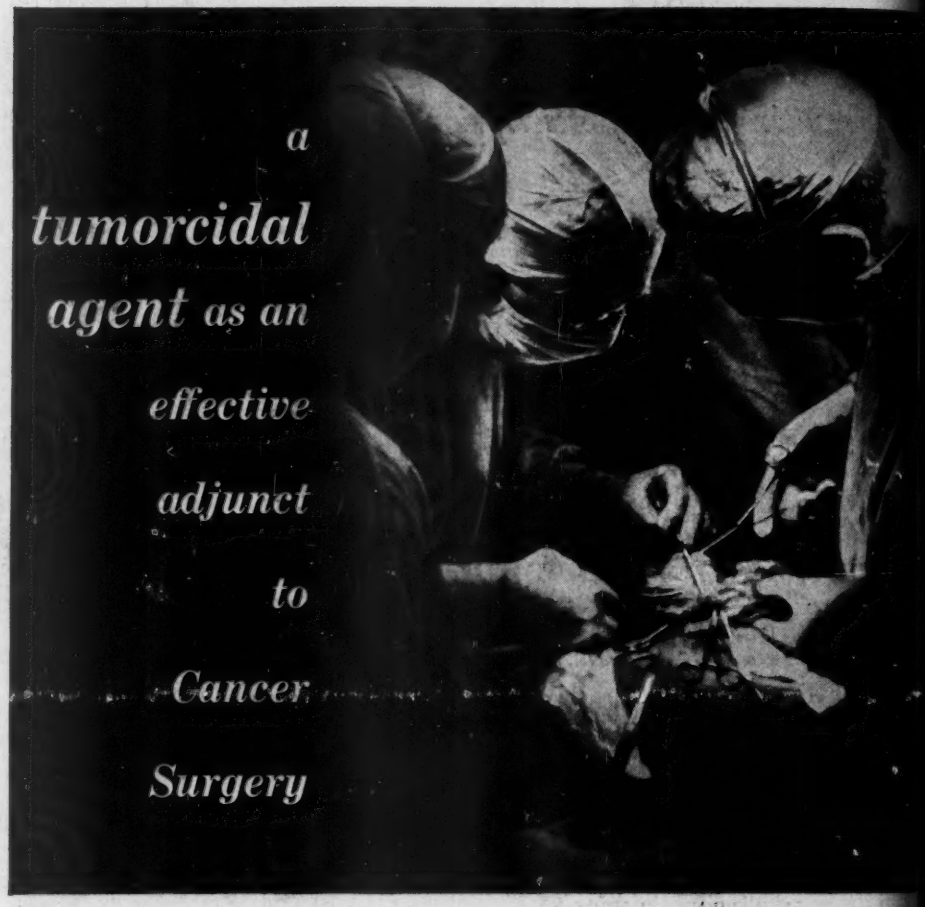

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